QUANTITATIVE GENETIC ANALYSIS OF A COMMERCIAL PIG POPULATION UNDERGOING SELECTION.

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Declaration

I hereby declare that this thesis is my own composition and is an account of analyses performed by me whilst studying for the degree of Doctor of Philosophy at the University of Edinburgh using data provided by the National Pig Development Company.

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September 1992.

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ABSTRACT

Data from a commercial pig population undergoing selection were analysed in order to estimate genetic variation and covariation. The data were collected between 1982 and 1988 and consisted of performance test records (food conversion ratio (FCR), total weight gain, average daily gain (ADG) and average daily food intake (ADFI) on test and four ultrasonic backfat measurements at shoulder, loin, C and K positions) for boars and gilts plus reproduction records (total number born, number born alive (NBA), litter weight, average piglet weight (PWt) and gestation length) on sows. Landrace and Large White breeds were subject to the same environment. The test regime varied between sexes; gilts were housed in groups, boars in pairs and gilts were fed on a lower feed scale than their boar contemporaries. During the data collection period the test regime changed for boars and gilts, leaving distinct groups of animals tested under different regimes (Test Management Groups or TMGs).

All analyses presented were performed using Individual Animal Model methods, which take account of all known relationships and selection on the trait, or traits, in the analysis.

Univariate heritability estimates for performance test traits were slightly lower on average than literature estimates. Differences in variance component and parameter estimates were observed between TMGs which could be explained by changes in feed regime, health status and halothane status; effects of selection were confounded with these.

Univariate heritability and repeatability estimates for reproduction traits were consistent with the available literature estimates.

Univariate analyses produced no evidence of maternal genetic variation for performance test or reproduction traits, while significant but generally low estimates of common litter of birth effects were detected for some performance test traits.

Correlations between performance test traits were estimated using univariate analysis of traits and linear combinations of traits. Genetic correlations between backfat depths and weight gain were negative for boars and gilts on a low feeding scale, although the gilt estimates were of greater magnitude consistent with gilts being more restricted. In TMGs where the feeding scale had been increased, the genetic correlations between backfat depths and weight gain were positive in boars and negative, but of smaller magnitude in gilts.

A method of analysis for bivariate traits was developed that used existing univariate programs. This was used to estimate correlations between performance test traits as recorded in males and females, and to estimate correlations between performance test and reproduction traits.

Genetic correlations between the sexes for performance traits were often close to unity indicating that there was little genotype-environment interaction between the sexes for nearly all traits across TMGs and breeds. All estimates of the genetic correlations between sexes exceeded 0.6 with the exception of two in the Large White; that for ADFI in the period when food restriction in gilts was greatest and that for FCR in the period when health status was low.

Performance test data combined across TMGs and all reproduction data were used to estimate the genetic correlations of ADG, C backfat depth

measurement, ADFI and FCR with PWt and NBA. Genetic correlations ranged from -0.029 to 0.082 with standard errors between 0.11 and 0.15. These estimates indicate that breeding value estimation can be carried out separately for the two groups of traits and selection on one group of traits only will have little effect upon the other group.

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CHAPTER 1.

INTRODUCTION.

The principle aim of this project was the estimation of genetic correlations between performance test and reproduction traits. In order to achieve this the analysis was developed from univariate analyses assessing different random effect models to estimation of correlations between performance test traits and onto bivariate analyses between pairs of performance test and reproduction traits.

This thesis reports a series of analyses to estimate genetic parameters for large commercial nucleus populations of Large White and Landrace pigs undergoing long term selection on an index of performance test traits. Despite the presence of many genetic parameter estimates for both performance test and reproduction traits in the literature new studies in this area remain relevant. This is because of advances in the design of algorithms and increased computer power which allow us to fit models which better account for the population structure than those previously used, using methods which take some account of the effects of inbreeding and selection upon parameter estimates. In turn these advances enable us to estimate parameters which past studies have omitted or estimated poorly. The genetic correlations between performance test and reproduction traits were of particular interest in this study.

Thompson (1989) gave four reasons why heritabilities, correlations, additive genetic variances and covariances are of interest. These were:-

(a) as a quantitative summary of inheritance in traits;

(b) in deciding whether selection will be effective (the low heritability of

litter traits in pigs has often been cited as one of the main reasons for their omission from selection criteria);

(c) in considering alternative selection schemes;

(d) in optimisation of a selection scheme.

In considering point (d) further, Thompson (1989) commented on optimisation of both population structure and incorporation of information from relatives and other traits using selection indices or Best Linear Unbiased Prediction (BLUP).

The selection index approach has been used for many years in the pig breeding industry. The traits included in the index have been performance test recorded traits such as growth rate, food conversion ratio and carcass traits. Although litter traits are of economic importance they have been subject to little attention in commercial breeding schemes (Haley *et al.*, 1986).

Avalos and Smith (1987) considered a number of possible methods of selecting for increased litter size, and demonstrated that by inclusion of information on relatives annual genetic responses of up to 0.5 pigs per litter could be achieved. BLUP methodology makes optimum use of information on relatives and so the inclusion of reproduction traits in the selection goal should become more practicable with the utilisation of these methods by pig breeders (assuming that the increased computer demands caused by their inclusion are not restrictive). This overcomes the obstacles to selection for reproduction traits posed by the inability to measure the traits in males or immature females.

BLUP procedures have been used for some time in the pig breeding

industry in countries where there is a national or regional testing and AI system in operation. Only recently have the commercial breeding companies in Britain become interested in the use of BLUP within their closed pyramid structure (Webb and Bampton, 1988). The reasons for this delay are the expected increase in inbreeding resulting from a tendency to select related animals and the limitations of computer facilities and algorithms. However, the inbreeding effect can be controlled by limiting the numbers of full and half sibs selected, while the size of the population undergoing selection can be increased in order to maintain selection pressure by creating genetic links between herds within a breeding pyramid using AI (Webb and Bampton, 1988) or between pyramids.

In order that true multivariate BLUP can be performed it is required that all parameters are known without error. While this can never be achieved, estimates of the parameters can be produced. Towards this end, a series of Individual Animal Model (IAM) Restricted Maximum Likelihood (REML) analyses were performed and are presented in the chapters which follow.

Both the IAM and REML have been the subject of much research effort in recent years. Consequently, as well as there being many research papers available on the two subjects, there are also a number of up to date reviews in the literature. For reviews of the IAM see Kennedy *et al.* (1988, concentrates on the genetic properties of animal models) and Hill and Meyer (1988, this contains both theoretical and practical considerations for the use of animal models for both BLUP and REML).

In principle the IAM is very straightforward, each animal's record being described in terms of its own genotype, rather than as the sum of the sire and dam components as in the previously most common model of analysis,

the hierarchical sire model. For example, an IAM for a trait with a common environmental effect (e.g. a growth trait of pigs with full sibs sharing a common environment before weaning) could be written as:-

$$Y_{ijk} = \mu + \beta_i + a_i + c_j + e_{ijk};$$

where μ is the overall mean, β_i is the combination of fixed effects for the i^{th} animal, a_i is the additive genetic effect of animal i, c_j is the j^{th} common environment effect and e_{ijk} is the random error associated with that record.

Meyer (1989b, 1990) has thoroughly reviewed the estimation of genetic parameters with particular emphasis on REML procedures, these having become the method of choice in recent years for the following reasons:-

a. in common with true maximum likelihood procedures, REML makes optimal use of all available information (Harville, 1977);

b. REML and ML estimators are frequently less biased by selection than ANOVA estimators (Meyer and Thompson, 1984, Sorensen and Kennedy, 1984, van der Werf and de Boer, 1990);

c. Unlike ML estimation, REML takes account of the loss of degrees of freedom from the fitting of fixed effects (Patterson and Thompson, 1971).

IAM REML procedures have further desirable properties in that all known relationships are accounted for and the models are easily extended to allow estimation of additional random effects (Meyer, 1989a).

The adoption of REML prodedures for variance component estimation has been aided by the extension to multivariate analysis (Thompson, 1973, Meyer, 1985, Meyer, 1991) and more recently the availability of derivative free algorithms which do not require the inversion of a large coefficient

matrix (Graser *et al.*, 1987, Meyer, 1988, 1989a and 1991).

The estimation of correlations between traits using a REML procedure is computationally demanding, and since its proposal (Thompson, 1973) has been limited to small data sets. The development of methodology to enable covariance estimation in special cases such as where design matrices are equal (Meyer, 1985, Thompson and Hill, 1990) or where two groups of related animals have records on two different traits (i.e. there is no environmental correlation between the traits, Schaeffer *et al.*, 1978, Juga and Thompson, 1990) has maintained interest and provided a platform for the development of more general multivariate algorithms (Meyer, 1991).

Estimates of genetic parameters

Parameter estimates for growth and reproduction traits can be found in recent reviews by Gu (1988) and Haley *et al.* (1988), therefore only recently published parameter estimates are reproduced here. Both of these reviews also include estimates of the genetic correlations between growth and reproduction traits, but since this is the main area of interest of this project and there have been so few estimates of these parameters they will be included here also.

In the review of Gu (1988) heritability estimates ranging from 0.20 to 0.76, 0.12 to 0.57 and 0.06 to 0.94 were reported for daily gain, food conversion ratio and backfat thickness, respectively. This variation in the parameter estimates for growth traits can also be seen in the review of Hutchens and Hintz (1981, quoted by Kaplon *et al.*, 1991) where mean literature estimates of the heritabilities for average daily gain and backfat were 0.38 and 0.39, respectively (Hutchens and Hintz, 1981, quoted by

Bates and Buchanan, 1988). In table 1.1 heritabilities for average daily gain and backfat thickness range from 0.14 to 0.52 and from 0.28 to 0.50, respectively. Weighted means of those heritability estimates with available standard errors were calculated as 0.25 and 0.43, using the inverse of the variance of the estimate as the weight (Haley *et al.*, 1988), thus:

$$\bar{h}^2_w = \sum_{i=1}^{n} h^2_i/var(h^2_i) \; / \; \sum_{i=1}^{n} 1/var(h^2_i)$$

where $\bar{h}^2_w$ is the weighted mean heritability of the n heritablity estimates in table 1.1 with available standard errors, h^2_i is the ith heritability estimate and $var(h^2_i)$ is the sampling variance of h^2_i; estimated as the square of the standard error.

Table 1.1 also contains estimates of the genetic correlation between average daily gain (ADG) and backfat depth (BF).

Comparison of parameter estimates under different feeding regimes from table 1.1 reveals little. The range of heritability estimates for ADG under *ad libitum* and restricted feeding are 0.11 to 0.50 and 0.18 to 0.44, respectively, while for BF they are 0.32 to 0.63 and 0.28 to 0.43. It may be expected that differences in the genetic variance of performance traits will be observed under *ad libitum* and restricted feeding, for example Cameron *et al.* (1988) hypothesised that less genetic variation for BF was observed under restricted feeding due to suppression of the pig's ability to express their genetic potential for the trait, although the effect of this upon parameter estimates may also be confounded with changes in the residual variance.

Table 1.1: Summary of recently published estimates of genetic parameters (h^2 and r_g) for average daily gain (ADG) and backfat depth (BF).

Ref.	Anim.	Breed	No. of:		Anal.	Feeding	h^2 x 100		r_g x 100
			Sires	Offs.		regime	ADG	BF	
1	B	LR_S	167	4722	PHS	restricted	18 (04)	33^m	18^m
	G	LR_S	167	4160	PHS	restricted	36 (06)	43^m	-45^m
	B	LW_S	164	5386	PHS	restricted	42 (07)	30^m	-2^m
	G	LW_S	164	3887	PHS	restricted	34 (06)	28^m	-33^m
2	B	-	-	2075	IAM_r	-	19	42	14
3	B	H+D	45	1762	OP	-	52 (20)	43 (25)^p	44(14)
4	B	LW	3932	114347	PHS_r	-	27	29^a	25^a
5	G+B	LR+D	35	160	IAM_r	*ad lib.*	50 (18)	38^m	87
	G+B	LR+D	35	160	IAM_r	restricted	44 (19)	39^m	58^m
6	C+G	LR+D	38	4635	IAM_r	*ad lib.*	14 (04)	50 (08)	-
7	B	LW+LR	242	1735	PHS_r	*ad lib.*	46	32^m	1^m
	G	LW+LR	242	3802	PHS_r	restricted	-	43^m	-
8	B	-	-	-	PHS_r	*ad lib.*	30 (06)	37 (07)	19 (13)
9					PHS	*ad lib.*	11 (13)	63 (14)	38 (31)
10					PHS	*ad lib.*	25 (15)	38 (13)	-3 (33)

References:
1. Gu *et al.*, 1989a
2. Groeneveld, 1991
3. Bates and Buchanan, 1988
4. Kaplon *et al.*, 1991
5. Cameron, 1990
6. McLaren *et al.*, 1990
7. Cameron *et al.*, 1990
8. Van Steenbergen *et al.* , 1990
9. Bereskin, 1987
10. Bereskin and Steele, 1988

Animals:
B - Boars G - Gilts C - Castrates

Breeds:
LR - Landrace LW - Large White D - Duroc H - Hampshire
(subscript s denotes synthetic line derived from given breed)

Analysis:
PHS - Paternal half-sib
IAM - individual Animal Model
OP - Offspring-Parent
(subscript r denotes REML estimate)

Parameters:
^{m} - mean of parameters quoted for BF measured at different positions
^{a} - weighted mean of parameters estimated in different groups
^{p} - probe BF

From table 1.1 it can be concluded that under *ad libitum* feeding, the correlation between ADG and BF is positive. That is, faster growing pigs produce more fat.

The situation under restricted feeding is less clear. Lean muscle deposition requires less energy than fat deposition, and so under restricted feeding it is expected that the faster growing animals would be those that were genetically predisposed to lean production, producing a negative genetic correlation between ADG and BF under these circumstances. Kielanowski (1968) and Fowler, Bichard and Pease (1976) used this to argue that selection for ADG under restricted feeding would identify those animals which were producing lean, and hence a correlated reduction in BF would be observed. However, a negative genetic correlation is not observed in all of the studies on restricted populations for which results are given in table 1.1, with estimates ranging from -0.45 to 0.58. This may be because of differences in the degree of restriction applied in each of these populations.

Cameron *et al.* (1990) and Van Steenbergen *et al.* (1990) give heritability and genetic correlation estimates for a variety of traits which included FCR and average daily food intake (ADFI) for boars fed *ad libitum*. These results are reproduced in Table 1.2.

Recently published estimates of the heritability of number born, number born alive and litter weight are given in table 1.3. From these, weighted means of 0.09, 0.10 and 0.13 were calculated for the three traits. Summarising earlier results, Haley *et al.* (1988) concluded that the heritability of litter size was 0.10.

Table 1.2: Estimates of heritabilities and genetic correlations taken from Cameron *et al.* (1990, ref. 1) and Van Steenbergen *et al.* (1990, ref. 2). h^2 x100 on diagonal, r_g x100 off diagonal and available standard errors in brackets.

Trait:	Ref.	Trait: ADG	BF	FCR	ADFI
ADG	1	46			
	2	30 (6)			
BF	1	1	32		
	2	19 (13)	37 (7)		
FCR	1	-60	47	36	
	2	-58 (11)	18 (15)	31 (7)	
ADFI	1	58	52	30	34
	2	57 (12)	39 (15)	33 (17)	24 (6)

Repeated measurements on a sow for reproduction traits are possible, and there may be a permanent environmental effect associated with a sow. The permanent environmental variance can then be estimated and the repeatability of a trait calculated as $(\sigma_a^2 + \sigma_c^2)/\sigma_p^2$; where σ_a^2, σ_c^2, and σ_p^2 are the additive genetic, permanent environmental and phenotypic variances, respectively (Falconer, 1981). In fitting this "repeatability" model it is assumed that the genetic correlation between successive measurements is unity. Haley *et al.* (1988) concluded from results of analyses of a number of large populations that there was little evidence to refute this, given that the available estimates of the genetic correlations between litter size as recorded in different parities would be biased by culling on performance in previous parities. The repeatability of litter size was given by Haley *et al.* (1988) as 0.15.

Table 1.4 contains estimates of the genetic correlations between performance test and reproduction traits. These estimates are inconsistent across analyses, with both sign and magnitude changing. For example, the

valid ($-1 \leq r_g \leq +1$) estimates of the genetic correlations of total number born with performance test traits range from -0.15 to 0.44 for average daily gain, and from -0.54 to 0.62 for backfat depth. Standard errors of the estimates (where available) are also high.

Although it is not possible to draw firm conclusions about the existence of non-zero genetic correlations between performance and reproduction traits from the estimates in the literature, a number of studies have reported changes in litter traits as a result of selection on performance traits. These changes have generally been antagonistic, for example Nelson *et al.* (1990) observed a reduction in the number born alive per litter in sows selected for extremely low backfat when compared to sows compared for extremely high backfat. De Nise *et al.* (1983) also reported a negative correlated response in litter size at birth and litter weight in an experiment where selection was on weight of lean cuts at a constant age.

Berruecos *et al.* (1970) also reported reduced litter sizes in a line selected for reduced backfat. However, Hetzer and Miller (1970) and Fredeen and Mikami (1986) observed no significant change in the litter size born. In these last two studies, however, the low lines were not as extreme for backfat depth as those of Nelson *et al.* and Berruecos *et al.*.

Selection experiments where selection was on an index to increase ADG and reduce BF have generally reported little change in the litter traits over generations (Cleveland *et al.*, 1988; Fredeen and Mikami, 1986) and Garnett and Rahnefeld (1976) reported no significant change in litter traits as a result of selection for increased ADG.

Table 1.3: Summary of recently published estimates of the heritability of total numbers born (NB), number born alive (NBA) and total litter weight (LWt). Heritabilities are given as a percentage of the total phenotypic variance, available standard errors in brackets.

Ref.	Breed	Litter records	Parities	Anal.	Heritability NB	NBA	LWt
1	LR_s	1018	pooled	IAM_r	11 (04)	07 (04)	12 (04)
	LW_s	863	pooled	IAM_r	10 (05)	12 (04)	14 (04)
2	Y	33365	pooled	PHSq		13	
3	Y	2960	first	IAM_r	13 (04)	13 (05)	
	LR	4225	first	IAM_r	13 (04)	09 (03)	
4	LW	41080	pooled	PHS_r		07	
5	Y	-	pooled	IAM_r		12	

References:
1. Gu *et al.*, 1989b
2. McCarter *et al.*, 1987
3. Southwood and Kennedy, 1990
4. Kaplon *et al.*, 1991
5. Sorensen, 1990

Breeds:
LR - Landrace LW - Large White Y - Yorkshire
(subscript s denotes synthetic line derived from given breed)

Analysis:
PHS - Paternal half-sib IAM - individual Animal Model
(subscript r denotes REML)

Table 1.4: Summary of published estimates of the genetic correlations between two performance test traits (ADG and BF) and three reproduction traits (NB, NBA and LWt). Correlations are presented as r_gx100, available standard errors in brackets.

Test Trait:			ADG			BF		
Reprod. Trait:			NB	NBA	LWt	NB	NBA	LWt
Ref.	Brd	Par						
1	-	1-4	6	-	-	-	-	-
2	D	1,2[a]	-	-	-	-21	-	-
	Y	1,2[a]	-	-	-	62	-	-
3	LW	-	-8	-	-	11	-	-
4	LW	1	16	-	-	-31[m]	-	-
	LW	2	-2	-	-	-32[m]	-	-
	LR	1	-15	-	-	-24[m]	-	-
	LR	2	44	-	-	-101[m]	-	-
5	-	1,2[a]	11(42)	-7(44)	45(26)	-32(30)	-21(33)	-15(25)
6	LR	1	-	-	-	17	13	-
	LR	2	-	-	-	-8	-5	-
	Y	1	-	-	-	24	22	-
	Y	2	-	-	-	18	13	-
7	-	1	-214(96)	-	-20(18)	-54(250)	-	-66(19)
8	LR	1	-	-	-	3	-	-
	LR	1	-	-	-	51	-	-

References:

1. Vogt*et al.*, 1963
2. Hetzer and Miller, 1970
3. Legault, 1971
4. Morris, 1975
5. Vangen, 1980
6. Johansson and Kennedy, 1983
7. Bereskin, 1984
8. Löbke *et al.*, 1986

Breeds:

LR - Landrace LW - Large White Y - Yorkshire D - Duroc

The results of these selection experiments may lead us to conclude that there is negative genetic correlation of backfat depth with litter traits and that this correlation may be increased in magnitude as backfat depth becomes more extreme as a result of selection. If so, it would not be expected that large changes would have been observed in the index selection experiments, since the selection pressure on BF is reduced compared to single trait selection. However, in the long term the continued selection on an index designed to reduce backfat depth may generate genetic correlations between performance test and reproduction traits which may be significant. Consequently, the reappraisal of the genetic correlation estimates is required in order that any modifications required to the selection procedure can be carried out, for example changing selection index weights to maintain a given level of backfat depth, or inclusion of litter traits in the selection index.

Maternal effects

Falconer (1960 and 1965) modelled a maternal effect of the size of litter into which a female was born on the size of litter she subsequently produced, in order that a negative maternal effect of litter size at birth could be explained in mice. That is, mice from large litters produced small litters and vice versa. This model need not be limited to the effect of litter size, any differences between dams at the genotypic level can be phenotypically expressed in their offsprings' performance for a given trait.

Willham (1963) derived equations for the covariances between different groups of relatives, showing the bias that would exist in estimates of the heritability from daughter-dam regression as opposed to paternal

half-sib estimation.

A number of studies of the maternal effect of litter size of birth or rearing on subsequent litter size performance have been carried out. Revelle and Robison (1973) concluded that there was a significant negative maternal effect on litter size from three pieces of evidence: gilts born in small litters produced larger litters than those born in large litters, the daughter-dam estimate of the heritability was less than half that of the granddaughter-granddam estimate and the average daughter-dam regression within groups of dams with small, average and large litter size were positive, around zero and negative, respectively. Studies designed to measure the size of this maternal effect by standardising rearing litter sizes after birth (Van der Steen, 1985; Rutledge, 1980) confirmed this result.

From an analysis of litter size data from a large commercial breeding company population, Avalos (1985) concluded that maternal effects on litter size are negligible where cross fostering to reduce the variance in the size of litter reared is regularly practised. Haley *et al.* (1986) reviewed a number of studies and came to a similar conclusion.

Southwood and Kennedy (1990) produced estimates of additive maternal heritabilities for number born, number born alive and number weaned of between 0 and 0.08 (with additive direct heritabilities between 0.06 and 0.13) for Landrace and Yorkshire gilts.

Maternal effects of size of litter of birth on growth and carcass traits have been reported by Standal (1973) and Willeke and Richter (1979).

Löbke *et al.* (1986) reported differences between paternal half sib estimates of genetic correlations and daughter-dam regression estimates

which may have been due to maternal effects. Maternal effects are assessed for performance test and reproduction traits in chapters 2 and 5, respectively, of this thesis.

Genotype environment interactions

The pig breeding industry is generally organised in a breeding pyramid structure. That is, selection is carried out in a nucleus and genetic progress is disseminated to the lower levels. In this way, resources can be concentrated in the nucleus where expensive performance testing is performed.

Genotype environment interactions (GxE) may arise both within and between levels of a breeding pyramid (Brascamp *et al.*, 1985). Falconer (1952) showed that GxE were the result of a change in the ranking of genotypes in different environments, resulting in genetic correlation estimates of less than one between a trait as recorded in two different environments. The estimation of the genetic correlation is the standard way of investigating GxE.

The effect of GxE is a reduced rate of genetic gain. If performance in a production system environment is the goal, then if the genetic correlation between this and performance in the selection environment is not unity maximum genetic gain will not be achieved. Similarly, within the nucleus, if groups of animals are subject to different environments the ranking of the genotypes may not be the same across the environments.

Webb and Curran (1986) reviewed genotype x environment interaction, with particular reference to interaction between selection regime and production system, and identified a number of possible causes of GxE.

These causes also apply within a pyramid level. Included in this list were differences in emphasis placed on traits in the selection objective, housing (number of pigs per pen), diet and feeding level, parity of dam, sex, health status and degree of environmental control. In practice, many of these factors will be confounded; for example, gilts on test are often housed and fed differently from boars on test and subject to a different selection index.

In the present study, performance test records collected over a six year period were analysed. Boars and gilts were subjected to different test regimes and as part of the study the genetic correlations between traits as recorded in boars and gilts were estimated. These were used to decide whether the male and female performance test data sets could be combined, assuming no genotype-sex interaction, for use in the analysis to estimate genetic correlations between performance test and reproduction records. The analysis estimating genetic correlations for performance test traits between the sexes is detailed in chapter 3.

CHAPTER 2.

UNIVARIATE INDIVIDUAL ANIMAL MODEL ESTIMATES OF GENETIC PARAMETERS FOR PERFORMANCE TEST TRAITS OF LANDRACE AND LARGE WHITE PIGS TESTED IN A COMMERCIAL NUCLEUS HERD.

INTRODUCTION

Genetic parameter estimates are dependent upon the model used to describe the data and the assumptions inherent in the chosen method of analysis. Most of the estimates of parameters found in the literature were obtained using Least-Squares procedures based either on covariation between full and half sibs or on offspring-parent regression, largely due to a lack of computing resources. It is frequently assumed that dams are completely nested within sires and the population is unselected. These types of analysis are not best suited to estimation of parameters from commercial pig populations, in which selection has been carried out intensively over many generations and sows are mated to a number of boars during their lifespan. Individual Animal Model Restricted Maximum Likelihood (REML) techniques are capable of producing estimates of parameters which make full use of the population structure, using offspring-parent and sib information, and which are less biased by selection (Van der Werf and de Boer, 1990).

The Animal Model is conceptually easy to understand, and extension to estimate other hypothesised random effects as well as additive genetic effects is straightforward (Meyer,1989a). The random effects considered in this study are additive direct genetic, common environmental, additive

maternal genetic, and cytoplasmic genetic effects.

Evidence of additive maternal effects on performance test traits is limited. Standal (1973) and Willeke and Richter (1979) reported maternal effects for growth and carcass traits due to the size of the litter into which the animal was born and parity of the dam. Cytoplasmic effects were reported for carcass traits by Dzapo *et al.* (1983).

As a result of changes in market requirements, breeding goals of commercial pig producers have changed. This is reflected in changes in the management of herds and selection methods.

It is reasonable to assume that estimates of genetic and environmental parameters will be affected by these changes. This can be illustrated by considering the estimation of the heritability of growth rate from data sets collected in populations fed on restricted scale and *ad libitum* diets. When animals are fed on an *ad libitum* scale genetic variation in growth rate can be fully expressed. However, under restricted feeding the limitation imposed on energy intake will reduce both the genetic and residual variance in the population. As a result, heritability estimates for daily gain estimated from restricted populations may differ from those from *ad libitum* fed populations. In this study data collected over a six year period in a single nucleus herd during which the test regime was altered were analysed in groups according to the test regime and health status of the herd so that changes in the parameter estimates across these groups could be observed.

Parameters are population specific, variance component estimates being sensitive to environmental differences and population structure.

Therefore, for utilisation in breeding schemes, parameter estimates should be taken from the population in question, provided it has sufficient size and structure.

MATERIAL AND METHODS

Performance test records collected over a six year period on a single nucleus unit were provided by a large commercial breeding company. The records were on purebreeding Landrace and Large White boars and gilts under different management systems, the test management differing between the sexes and over time.

Test details:

The test can broadly be described as being over a weight range of 40-90kg (approximately 3-5 months of age), with an increasing feeding scale during the test. All animals finishing test in a given week were assigned to the same test batch, and during the analysis a test month was regarded as being a group of four consecutive test batches.

Total weight gain on test {WtG; recorded in kg} was calculated as weight at start of test minus weight at end of test, and from this the average daily gain on test {ADG; kg} was obtained. The food conversion ratio (FCR) was calculated as the ratio of avarage daily food intake to ADG, where average daily food intake (ADFI) was the total food intake during the test (measured in kg) divided by the number of days spent on test. Ultrasonic backfat depths were recorded at four points on each animal, a single measurement being taken at each point. Backfat depths were recorded in mm at the shoulder (Sh Fat), loin (L Fat) and approximate P2 and P3 positions (C Fat and K Fat). All traits were recorded on both sexes.

Gilts were housed in groups of ten and had a greater degree of restriction in the amount of food offered than boars, which were housed in pairs in a more controlled environment. Further details of the differences between test regimes appear in a subsequent section ('Division of data', figures 2.1-2.8 and table 2.1).

Data validation:

Each record contained pedigree identifiers of the animal and both parents, birth and test dates and various codes (e.g. test batch number) associated with the recorded variables. Because of the need for accurate pedigree information in forming the relationship matrix, the records were rigorously checked for recording errors.

Data recording errors within variables could only be identified if they resulted in values which were extreme for that variable, and resulted in the disposal of that record. By comparison of an individuals record with those of contemporaries the source of errors for some time period variables could be identified, and hence these records could be corrected.

Errors in the pedigree identifier of an individuals parents could be identified as they would be expected to produce parents with very few offspring or with an abnormal distribution of offspring births over time. Sires were expected to have a single, continuous period of offspring births and dams were expected to farrow approximately once every six months. Many errors of this type were corrected after comparisons were made among groups of contemporary records, the final decision as to the true identity being made manually.

Records containing errors were deleted where the source of the

apparent error could not be located and explained or where the sire of the record appeared only once in the data.

Division of data:

The data set was divided into breeds, previous studies having indicated that estimates of genetic parameters may differ between Landrace and Large White breeds. Within each breed the analyses were performed upon boars and gilts separately because of the differences in the performance test and selection index used.

From these four basic groups (LR♂, LR♀, LW♂ and LW♀), a number of smaller groups were formed, to take account of changes in the test management and health status of the herd. These changes were identified using the data on ADFI and number of days spent on test.

By using the maximum individual ADFI within a test month as an indicator of food allowance within that month (assuming that at least one animal in each month eats all the food offered to it), then longterm changes in feeding levels could be identified (within test month changes could not be differentiated from recording errors).

Figures 2.1-2.4 show graphs of maximum ADFI and variance of ADFI against test month (test month 1 is approximately May 1982) for the four breed/sex combinations. They show that there was:

- a consistent rise in the maximum ADFI in all breed/sex combinations around half way through the data collection period, which was explained by the company as an increase in the food allotment to allow greater expression of appetite by individuals;
- a period of haphazard decline in the maximum ADFI in all

breed/sex combinations during the last quarter of the data collection period, which was explained as a general loss of appetite by the pigs in the herd brought about by a decline in the health status of the unit;

- an earlier increase in the maximum ADFI in the data on Large White boars, consistent with an increase in the food allotment;

- a number of inconsistent changes in the maximum ADFI which could be due to trials being carried out by the company or data errors;

- greater variation in ADFI in males than in females; indicating that the males were less restricted than the females while on test;

- a marked decline in the variance in ADFI in gilts during the second quarter of the data collection period, which could be due to the effect of selecting for faster growth and hence bigger appetites (resulting in more animals eating all the food offered);

- an increase in the variation in ADFI associated with increasing the food allotment; this was most noticeable in the gilts which were more restricted previously.

On a fixed time period test all animals will start and finish test together, hence there will be no variation in number of days spent on test.However, on a fixed weight range test, slower growing animals will remain on test longer, causing variation in days on test amongst individuals finishing test in the same week. Figures 2.5-2.8 are graphs of means and variances of number of days spent on test against month of test. These were used to define the type of test (fixed time period or fixed weight range) undertaken at any time in the data collection period.

Figure 2.1: Graph of maximum ADFI and variance in ADFI within a 4 week period (test month) against test month for Landrace boars.

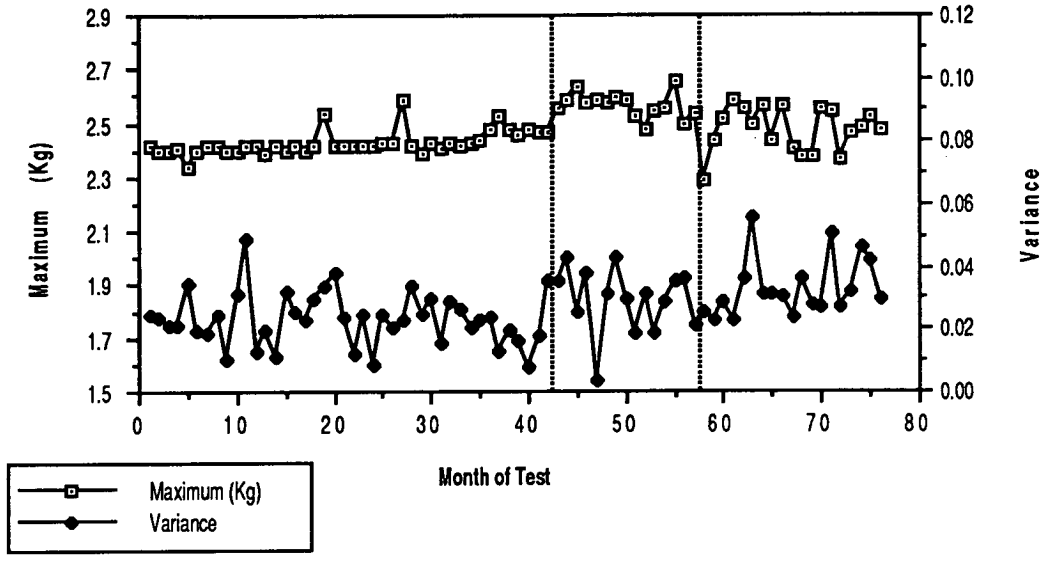


Figure 2.2: Graph of maximum ADFI and variance in ADFI within a 4 week period (test month) against test month for Landrace gilts.

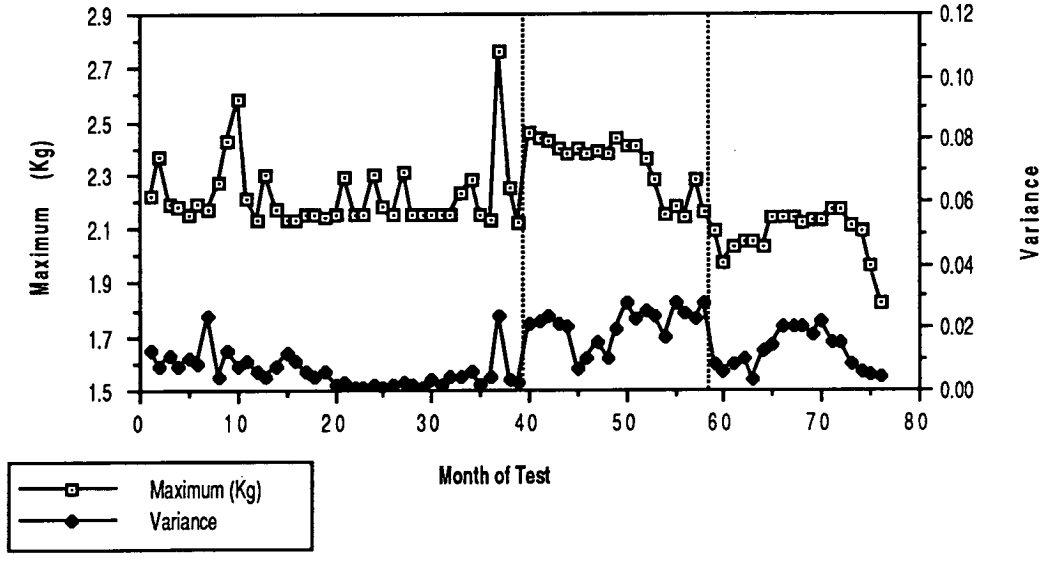


Figure 2.3: Graph of maximum ADFI and variance in ADFI within a 4 week period (test month) against test month for Large White boars.

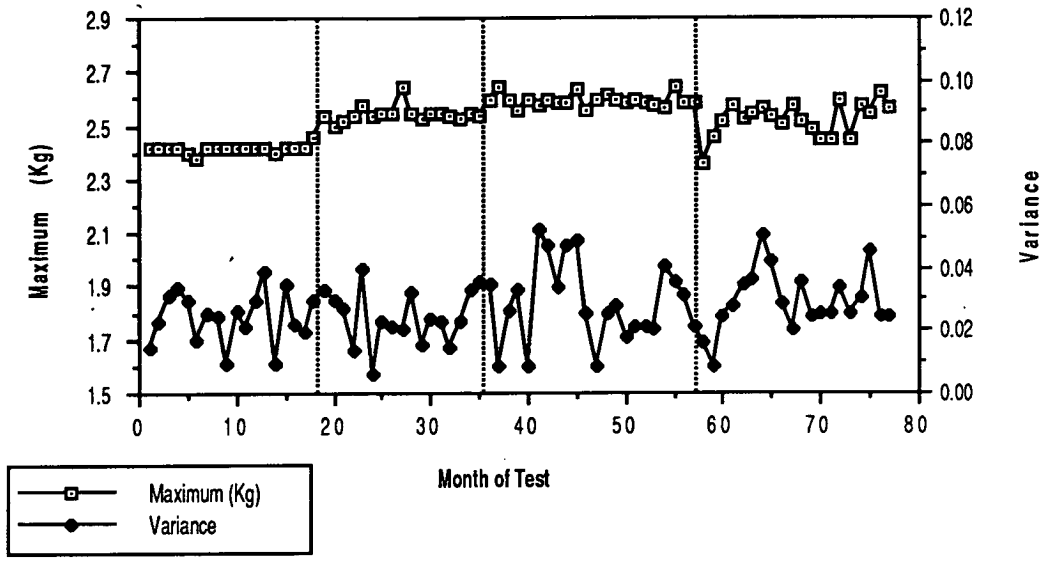


Figure 2.4: Graph of maximum ADFI and variance in ADFI within a 4 week period (test month) against test month for Large White gilts.

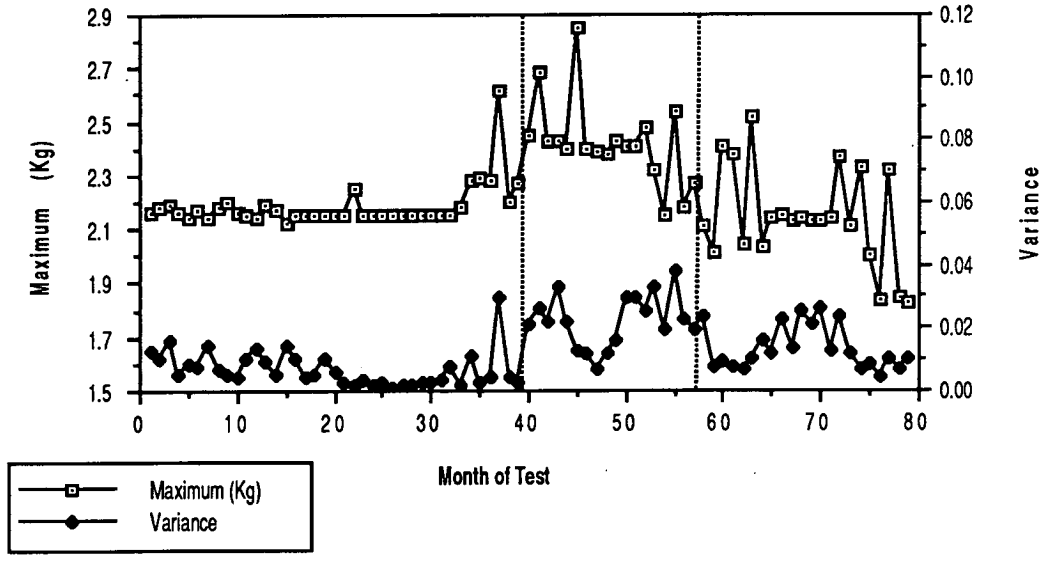


Figure 2.5: Graph of mean and variance in number of days spent on test against test month for Landrace boars.

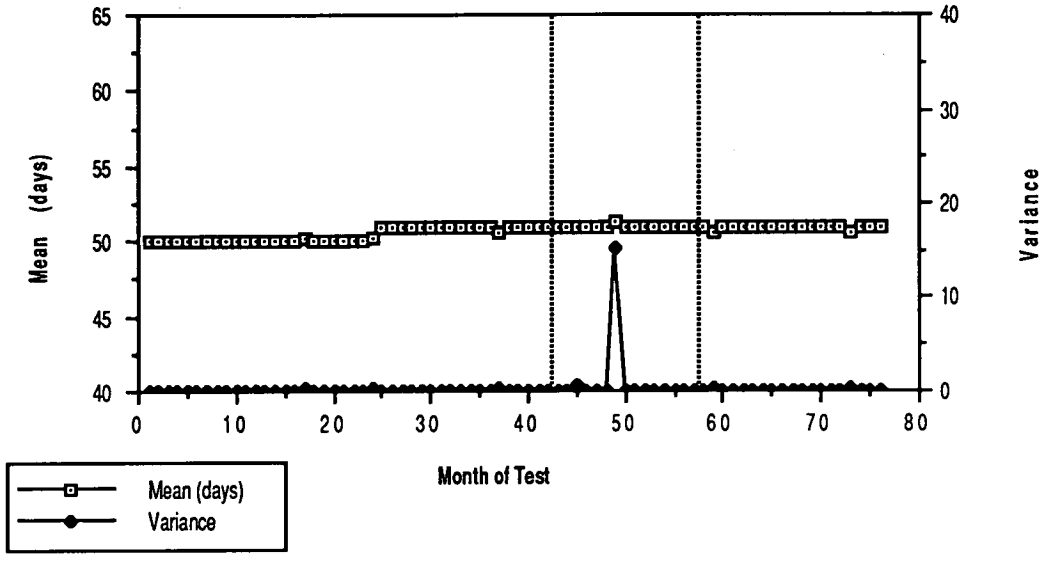


Figure 2.6: Graph of mean and variance in number of days spent on test against test month for Landrace gilts.

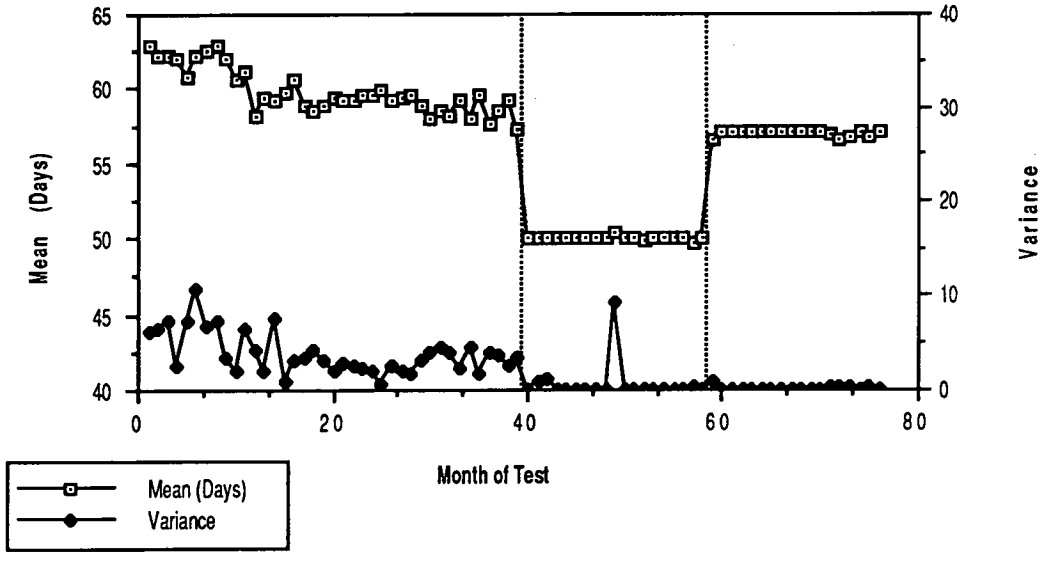


Figure 2.7: Graph of mean and variance in number of days spent on test against test month for Large White boars.

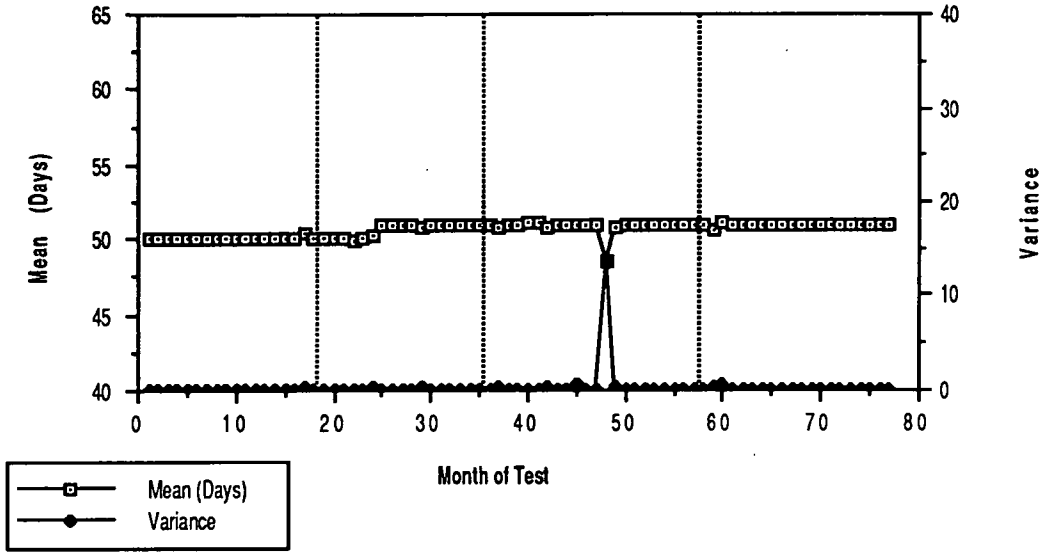


Figure 2.8: Graph of mean and variance in number of days spent on test against test month for Large White gilts.

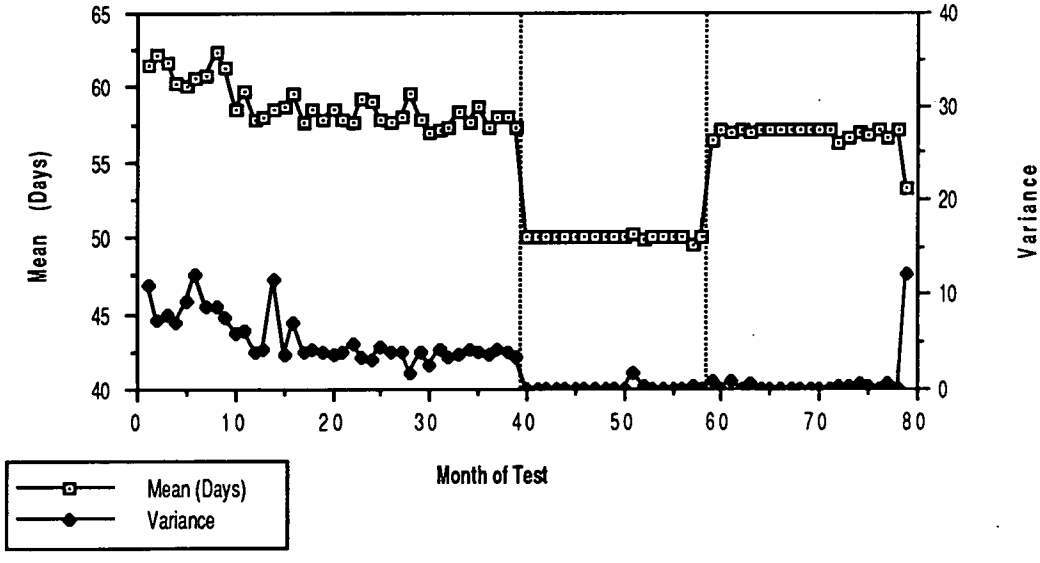


Table 2.1: Population structure summary for performance tested pigs; sub-divided into Test Management Groups.

Breed	Sex	TMG	Period	n_{rec}	n_{anim}	n_m	n_c	$c_{1\%}$	n_s	n_d	$d_{1\%}$
LR	♂	1	1-42	1607	2076	42	827	44	67	419	21
		2	43-57	538	915	15	279	45	30	190	26
		3	58-76	621	1052	19	313	41	28	198	25
		1-3	1-76	2766		76	1414	43	98	656	18
	♀	1	1-39	2798	3135	39	946	21	62	430	8
		2	40-57	1743	2158	18	462	11	36	256	7
		3	58-76	1654	2085	19	427	12	28	235	8
		1-3	1-76	6195		76	1821	16	99	732	6
	♂,♀	-	1-76	8961	9342	76	1937	9	99	752	4
LW	♂	1	1-18	873	1153	18	389	37	34	243	24
		2	19-35	780	1137	17	382	43	41	247	26
		3	36-57	1112	1615	22	482	34	49	271	26
		4	58-77	884	1411	20	391	32	35	242	20
		1-4	1-77	3649		77	1629	35	114	770	16
	♀	1	1-39	2690	3014	39	966	26	70	469	13
		2	40-55	1702	2183	16	445	12	42	270	10
		3	56-79	2100	2642	24	578	15	44	322	12
		1-3	1-79	6492		79	1966	19	127	873	12
	♂,♀	-	1-79	10141	10545	79	2123	10	131	907	9

Where: TMG - Test Management Group code (within breed and sex);
Period - given in test months;
n_{rec} - number of records in data set;
n_{anim} - number of animals (including those in pedigree only);
n_m - number of test months represented in data set;
n_c - number of levels of common litter of birth effect;
$c_{1\%}$ - percentage of levels of common litter of birth effect represented by a single record;
n_s - number of sires;
n_d - number of dams;
$d_{1\%}$ - percentage of dams represented by a single offspring.

From the graphs it can be seen that boars were tested using a fixed time period test throughout the whole of the recording period whilst the gilts were switched from a fixed weight range test to a fixed time period test at the same time as the food allocation was increased. Occasional blips in the graphs of days spent on test during fixed time testing periods can only be attributed to necessities of herd management and research, or uncorrected recording errors.

The vertical dotted lines on the graphs in figures 2.1-2.8 delimit the boundaries of the data groups as it was decided to analyse them. The population structure both within and across all of the test management groups is summarised in table 2.1.

Analyses performed:

Each data set was analysed using the univariate Derivative-Free REML (DFREML) algorithm developed by Dr. K. Meyer. This algorithm is an extension of the method proposed by Smith and Graser (1986, and Graser *et al.*,1987) to allow simultaneous estimation of up to three random effects (Meyer,1989a).

Making use of this capability it was possible to estimate a common environmental effect, which would be expected to affect littermates (or more precisely litter of birth mates since cross-fostering was widely practised in the population) and an effect of the maternal genotype. For a trait, i, which is affected by a second trait expressed in a relative, Willham (1963) showed how the genetic (co)variances of the two traits would be expected to be partitioned into the between relative genetic covariances for trait i. The additive maternal genetic effect can be viewed in this way, with the maternal

trait being the whole of the maternal genotype. By making use of the covariances between relatives the additive maternal genetic variance can be estimated just as for the additive direct genetic variance.

The statistical significance of the different random effect models was tested using a likelihood ratio test such that $-2(\ln L_1 - \ln L_2)$ has a Chi-squared distribution with n_2-n_1 degrees of freedom, where n_i is the number of random effects in model i and L_i is the maximum value of the likelihood function.

All data sets were analysed fitting the following Individual Animal models (models 1,2 and 3, respectively):

$$Y_{ijklm} = a_i + MT_j + b1.L_{ijk} + b2.W_{ijl} + b3.D_{ijm} + e_{ijklm}$$

$$Y_{ijklmn} = a_i + MT_j + b1.L_{ijkn} + b2.W_{ijln} + b3.D_{ijmn} + c_n + e_{ijklmn}$$

$$Y_{ijklmno} = a_i + MT_j + b1.L_{ijkno} + b2.W_{ijlno} + b3.D_{ijmno} + c_n + m_o + e_{ijklmno}$$

Where:

$Y_{ijklm}, Y_{ijklmn}, Y_{ijklmno}$ - are the phenotypic record of individual i for the trait under models 1,2 and 3, respectively;

a_i - is the random additive genetic effect of the i^{th} individual;

MT_j - is the fixed effect associated with the j^{th} month of test;

$b1.L_{ijk}, b1.L_{ijkn}, b1.L_{ijkno}$ - are the partial regressions of Y on length of test period (days) under models 1,2 and 3, respectively;

$b2.W_{ijl}, b2.W_{ijln}, b2.W_{ijlno}$ - are the partial regressions of Y on weight at start of test (kg) under models 1,2 and 3, respectively;

$b3.D_{ijm}, b3.D_{ijmn}, b3.D_{ijmno}$ - are the partial regressions of Y on age at start of test (days) under models 1,2 and 3, respectively;

c_n - is the random effect of the n^{th} litter of birth;

m_o - is the random additive genetic effect of the o^{th} dam;

$e_{ijklm}, e_{ijklmn}, e_{ijklmno}$ - are the random environmental effects associated with the records, Y_{ijklm}, Y_{ijklmn} and $Y_{ijklmno}$, respectively.

The expectations of the additive direct genetic effect (a_i), additive maternal genetic effect (m_o), litter of birth effect (c_n) and residual error effect ($e_{i.}$) are zero, and their variances are σ^2_a, σ^2_m, σ^2_c and σ^2_e respectively. The covariance between additive direct and additive maternal effects is σ_{am} and the covariances between all other combinations of random effects are zero.

Alternatively, the linear model can be written in matrix notation as:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e}$$

Where:

Y - is a vector of n_{rec} observations;

b - is a vector of n_f (where: $n_f=n_m+3$; n_m as in table 2.1, plus 3 covariates) fixed effects;

X - is an n_{rec} x n_f incidence matrix relating fixed effects to records;

u - is a vector of n_u random effects;

Z - is an n_{rec} x n_u incidence matrix relating random effects to records.

The following variance structure was assumed throughout:

$V(\mathbf{u}) = \mathbf{G};$

$V(\mathbf{e}) = \mathbf{R} = \sigma_e^2\mathbf{I};$

$cov(\mathbf{u},\mathbf{e}') = 0;$

$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}.$

In terms of the three models fitted here, we can see that **b** contains months of test and covariates of age at start of test, weight at start of test and number of days spent on test, and that within a test management group this remains constant for analyses under all three models. Similarly, the incidence matrix **X**, relating fixed effects to test records, remains unaltered.

Changes between the three models were in terms of the random effects fitted. Hence, the vector **u** and incidence matrix **Z** changed to accomodate this. The incidence matrix **Z** became block-diagonal when more than one random effect was included, each block consisting of the incidence matrix for one of the random effects. The changes made to the random effects vector, and to the assumed variance-covariance matrix **G** are summarised in Table 2.2.

In each Test Management Group the numerator relationship matrix, **A**, was calculated from pedigree information from all recorded animals within that group, and all pedigree information traced back through parental lines to the start of the data collection period. In this way all known relationships between recorded animals were taken account of, and pedigree information on animals without records and without direct paternal or

maternal links to animals in the data set was omitted.

Table 2.2: Summary of differences between models fitted.

Model:	1	2	3
Parameters:	h^2	h^2, c^2	h^2, m^2, σ_{am}/σ_p^2, c^2
Variance components:	σ_a^2, σ_e^2	σ_a^2, σ_c^2, σ_e^2	σ_a^2, σ_m^2, σ_{am}, σ_c^2, σ_e^2
u'	**a'**	**(a' c')**	**(a' m' c')**
G	$\sigma_a^2\mathbf{A}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \sigma_{am}\mathbf{A} & \mathbf{0} \\ \sigma_{am}\mathbf{A} & \sigma_m^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$

Where:

A - is the numerator relationship matrix;

a - is a vector of n_{anim} direct additive genetic effects, $\mathbf{a} \sim N(0, \sigma_a^2\mathbf{A})$;

m - is a vector of n_{anim} maternal additive genetic effects, $\mathbf{m} \sim N(0, \sigma_m^2\mathbf{A})$;

c - is a vector of n_c common environmental (litter of birth) effects, $\mathbf{c} \sim N(0, \sigma_c^2\mathbf{I})$;

h^2 - is the heritability;

m^2 - is the maternal genetic variance, σ_m^2, expressed as a proportion of the phenotypic variance, σ_p^2;

c^2 - is the common environmental variance, σ_c^2, expressed as a proportion of the phenotypic variance, σ_p^2;

$\sigma_{am}\mathbf{A}$ - is the covariance between **a** and **m'**

Standard error estimation

Smith and Graser (1986) showed how approximate standard errors of

DFREML estimates could be calculated by fitting a quadratic approximation to the likelihood surface around the maxima, thus:

$$q_0 + \sigma' q_1 + \sigma' \mathbf{Q} \sigma = L(\sigma)$$

Where: σ - vector of parameter estimates;

$L(\sigma)$ - Log likelihood evaluated at σ;

$\mathbf{Q}, q_0, q_1$ - are unknown.

Then the variance-covariance matrix of the maximum likelihood estimate of σ is approximately $(-2\mathbf{Q})^{-1}$. One feature of this procedure is that the estimates which are used in fitting the quadratic can be taken from the likelihood evaluations made during the analysis.

Unfortunately, this method proved to be inadequate for models containing more than one parameter (Meyer,1989a; Crump *et al.*, 1990). Sampling correlations between parameters produce likelihood surfaces which are not quadratic (Meyer,1990) and subsequently the estimate of $(-2\mathbf{Q})^{-1}$ is often found to be negative definite.

In the analysis of data collected in an experimental sheep population, Cameron and Bracken (1992) overcame this problem by considering each parameter in turn, fixing it at values around its maximum likelihood value and remaximising the likelihood function with respect to all remaining parameters. In this way they reduced the standard error calculations to a series of two-dimensional quadratic regressions, some account being taken of correlations among parameters when the likelihood function was remaximised.

The method used here involves taking each parameter in turn and fixing it at points around its maximum likelihood value and evaluating the

likelihood function at each of those points with all other parameters fixed at their maximum likelihood values. This method has an obvious advantage in the amount of computing time required over the method used by Cameron and Bracken (1992), one likelihood function evaluation being carried out at each point rather than a complete maximisation. However, the actual standard error estimates produced by Cameron and Bracken's (1992) method are expected to be better, having taken some account of sampling correlations between parameters.

There are two differences between the method used here and that of Cameron and Bracken (1992). If we consider the likelihood corresponding to the maximum likelihood estimates of all parameters as being $L_1(\gamma_1,\vartheta_{11}...\vartheta_{n1})$, where γ_1 is the maximum likelihood estimate of the residual variance and $\vartheta_{11}...\vartheta_{n1}$ are maximum likelihood estimates of the n parameters being estimated by the model. Then, in the estimation of the standard error on parameter 1, the likelihoods estimated at any fixed value of this parameter (point m) under either of the two methods considered are $L_m(\gamma_m,\vartheta_{2m}...\vartheta_{nm}|\vartheta_{1m})$ and $L_m(\gamma_m|\vartheta_{1m},\vartheta_{21}...\vartheta_{n1})$ for the methods of Cameron and Bracken (1992) and this thesis, respectively. If a likelihood ratio test comparing any two points for parameter 1 was carried out then the likelihood of Cameron and Bracken would provide a test of whether one value of parameter 1 differed from another, while the method of this thesis would test whether the two values differed given the values of all other parameters.

When fixing all parameters except the one of interest at their maximum likelihood value, no account is taken of the sampling correlations between these parameters and the parameter of interest. Given that Cameron and

Bracken (1992) observed little change from the maximum likelihood values of parameters when remaximising at points around the maxima for the parameter of interest, it may be expected that this would have little effect on the magnitude of the standard error estimates presented here.

In order to speed up the estimation process further, it is desirable to evaluate the likelihood function at as few points as possible. Limited test runs indicated that the use of three points (the maximum likelihood value of the parameter,P, and $P \pm \delta P$; where $\delta = 0.1$), gave the same estimated standard errors as runs involving more than one value of δ, to the degree of accuracy given here (2 decimal places).

Estimation of cytoplasmic effects.

The model proposed by Southwood *et al.* (1989) was utilised to estimate a random effect of cytoplasmic line upon the performance test data for the Landrace breed. The pedigrees of all animals were traced back to identify founder females and all animals were assigned to a cytoplasmic line on this basis.

Table 2.3: Summary of distribution of Landrace test records among cytopasmic lines.

TMG	♂			♀		
	n_{cyto}	$n_{r/cyto}$	$cyto_{1\%}$	n_{cyto}	$n_{r/cyto}$	$cyto_{1\%}$
1	259	6.20	19	274	10.21	7
2	78	6.90	13	104	16.76	7
3	82	7.57	15	90	23.17	6

Where: TMG - is the Test Management Group within sex;

n_{cyto} - is the number of cytoplasmic lines in this TMG;

$n_{r/cyto}$ - is the average number of records per cytoplasmic line;

$cyto_{1\%}$ - is the percentage of cytoplasmic lines represented by a single record.

A number of related assumptions are made in fitting this model. Each founder female was assumed to be from a unique cytoplasmic source. The cytoplasmic component was constant from one generation to the next, which implies that a constant number of copies is passed on each generation, no mutation is occurring, there is no alternative source of cytoplasmic DNA (e.g. the sire) and the pedigree is correct.

The model used was then equivalent to the previously fitted model 3 with cytoplasmic line effects substituted for common environmental effects such that the random effects vector **u'** is now (**a'**, **m'**, $\mathbf{c}'_{cyto}$), the same fixed effect structure being fitted to the data and with **G** now being equal to:

$$\begin{bmatrix} \sigma^2_a\mathbf{A} & \sigma_{am}\mathbf{A} & \mathbf{0} \\ \sigma_{am}\mathbf{A} & \sigma^2_m\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \sigma^2_{c_{cyto}}\mathbf{I} \end{bmatrix}$$

RESULTS

Phenotypic means of the traits are presented in table 2.4. Results from analyses under models 1, 2 and 3 (see table 2.2 for a summary of differences between these models) are given in tables 2.9.i to 2.9.xiii. The results which appear in table 2.9.i to 2.9.xiii are REML estimates of variance components as proportions of the total phenotypic variance, residual and phenotypic variances, log likelihoods and likelihood ratio test statistics. Abbreviated results, in the form of heritabilities, common litter of birth effects and residual variances for selected traits from analysis under model 2

(common litter of birth effect included, but no additive maternal effect), are given in tables 2.6 and 2.7.

Comparison of models containing combinations of additive genetic, litter of birth and additive maternal effects.

By comparing the test statistic $-2(\ln L_m - \ln L_n)$ (where: model n indicates the model containing the random effects to be tested, and model m is a model omitting these random effects) with the percentage points for the Chi-squared distribution (given in table 2.5), the statistical significance of including various combinations of random effects can be approximated.

Likelihood ratio tests were used to test the significance of the following random effect combinations:-

effects:	L_n	L_m	d.f.
common litter of birth	L_2	L_1	1
additive maternal additive x additive covariance	L_3	L_2	2
additive maternal additive x additive covariance cytoplasmic line	L_{cyto}	L_1	3

For a small number of analyses, the likelihood test statistic produced was negative, but of low magnitude, indicating that the maximum of the likelihood function had not been reached for the model being tested. This could have been avoided by lowering the value of the convergence criterion; but taking account of the small size of the negative test statistics and their infrequency, the increased computing involved in this was felt not to be justified.

Table 2.4: Means of traits across Test Management Groups.

Trait	Breed/Sex	Test Management Group:*			
		1_1		2_1	3_1
		1_2	2_2	3_2	4_2
Sh Fat (mm)	LR♂	28.03		27.80	26.78
	LR♀	30.04		28.82	27.79
	LW♂	30.01	30.06	30.41	29.40
	LW♀	30.90		31.57	29.82
L Fat (mm)	LR♂	12.58		12.91	12.40
	LR♀	14.09		13.26	12.88
	LW♂	13.00	13.86	13.54	13.21
	LW♀	14.34		14.28	13.47
C Fat (mm)	LR♂	10.69		10.31	9.52
	LR♀	11.51		9.99	9.52
	LW♂	11.22	12.09	11.12	10.53
	LW♀	12.00		11.19	10.33
K Fat (mm)	LR♂	11.01		10.60	9.73
	LR♀	11.79		10.25	9.74
	LW♂	11.56	12.41	11.43	10.80
	LW♀	12.31		11.52	10.60
ADFI (kg)	LR♂	2.20		2.30	2.14
	LR♀	2.08		2.16	1.93
	LW♂	2.21	2.31	2.35	2.22
	LW♀	2.06		2.21	1.92
WtG (kg)	LR♂	50.20		51.09	47.90
	LR♀	47.25		42.02	43.09
	LW♂	50.73	55.12	53.12	51.89
	LW♀	48.57		43.85	44.11
ADG (kg)	LR♂	1.00		1.00	0.94
	LR♀	0.79		0.84	0.76
	LW♂	1.01	1.09	1.05	1.02
	LW♀	0.83		0.88	0.79
FCR (kg Feed/ kg Gain)	LR♂	2.23		2.31	2.30
	LR♀	2.64		2.58	2.55
	LW♂	2.20	2.13	2.26	2.20
	LW♀	2.50		2.53	2.44

* - subscript 1 refers to TMG codes for LR♂, LR♀ or LW♀; and 2 to LW♂.

Table 2.5: Percentage points of the Chi-squared distribution (taken from Snedecor and Cochran, 1980)

d.f.	Probability of a greater value:		
	0.1	0.05	0.01
1	2.71	3.84	6.63
2	4.61	5.99	9.21
3	6.25	7.81	11.34

Inclusion of a common litter of birth effect produces significant likelihood test statistic results across traits and Test Management Groups in gilts of both breeds. In boar data sets fewer significant litter effect estimates were detected, and these were predominantly found in the first Test Management Groups which made up the first half of the data collection period.

Testing the significance of inclusion of the additive maternal genetic effect and the additive maternal - additive direct covariance in the model already containing a common litter of birth effect gave less clear results. There were a small number of analyses which suggested the presence of a significant effect ($p<0.1$) for a given trait, and with only one exception these were in the final Test Management Groups of Large White boars and gilts.

The test statistic used for the model containing the cytoplasmic line effect was a comparison of model 1 with the cytoplasmic model, such that the test was for a significant improvement in fit of the model due to the three effects m^2, c^2_{cyto} and σ_{am}/σ^2_p. Consequently, in the few analyses where the test statistic was significant, the magnitude of c^2_{cyto} was still low, indicating that the significance was due to the inclusion of the additive-maternal effect

rather than the cytoplasmic effect. This is born out by the relatively high value of the test statistic for the equivalent analyses under model 3.

Differences between breeds, sexes and TMGs:

REML estimates of variance components, and therefore parameters, were observed to be different across test management groups, breeds and sexes. These differences can be seen in tables 2.6 and 2.7, where averages across TMGs, within breed and sex (table 2.6), and across breed, within TMG and sex (table 2.7), are given. When making comparisons between the averages in tables 2.6 and 2.7, the different values used in constructing each of the means are assumed to be replicates.

From the across TMG averages in table 2.6 it can be seen that, generally, Large White estimates of heritability of performance test traits are greater than those from Landrace. Within a breed the following observations were made about the heritability:-

i. Estimates for ultrasonic backfat measures and FCR were greater from females than from males;

ii. Estimates from males were greater than those from females for ADFI;

iii. For ADG and WtG estimates were greater from males for the LW breed, while there was little difference between the estimates from either sex in LR.

Differences between breeds and sexes can also be observed for residual variance and common litter of birth effect estimates, again using the average estimates calculated across TMGs (table 2.6)

Table 2.6: Selected heritability and common litter of birth effect estimates across breeds and sexes. Model of analysis included additive direct and common litter of birth random effects only; results taken from tables 2.5.i - 2.5.xiii.

Trait:	Breed and Sex	σ_e^2 TMG:*				$\bar{\sigma}_e^2$	h^2 TMG:*				$\bar{h}^2$	c^2 TMG:*				$\bar{c}^2$
		1_1	1_2	2_2	2_1 3_2 3_1 4_2		1_1	1_2	2_2	2_1 3_2 3_1 4_2		1_1	1_2	2_2	2_1 3_2 3_1 4_2	
C Fat	LR♂	1.28			1.33 1.36	1.32	0.28			0.36 0.34	0.33	0.10			0.01 0.01	0.04
	LR♀	1.33			1.25 0.97	1.18	0.39			0.25 0.46	0.37	0.07			0.09 0.02	0.06
	LW♂	1.49		1.58	0.98 1.49	1.38	0.34		0.35	0.60 0.36	0.41	0.08		0.12	0.01 0.00	0.05
	LW♀	1.51			1.36 1.29	1.39	0.50			0.44 0.37	0.44	0.01			0.05 0.03	0.03
ADFI	LR♂	0.0143			0.0178 0.0226	0.0182	0.22			0.19 0.09	0.17	0.09			0.11 0.14	0.11
	LR♀	0.00467			0.0118 0.0105	0.0090	0.09			0.17 0.05	0.10	0.07			0.12 0.12	0.10
	LW♂	0.0179		0.0135	0.0172 0.0222	0.0177	0.10		0.31	0.29 0.14	0.21	0.10		0.11	0.02 0.05	0.07
	LW♀	0.00507			0.0149 0.0120	0.0107	0.12			0.14 0.07	0.11	0.11			0.06 0.04	0.07
ADG	LR♂	0.00582			0.00657 0.00828	0.00689	0.20			0.08 0.17	0.15	0.05			0.01 0.00	0.02
	LR♀	0.00222			0.00391 0.00403	0.00339	0.10			0.20 0.15	0.15	0.08			0.09 0.10	0.09
	LW♂	0.00629		0.00420	0.00541 0.00769	0.00590	0.23		0.41	0.36 0.21	0.30	0.07		0.06	0.03 0.01	0.04
	LW♀	0.00211			0.00433 0.00494	0.00379	0.17			0.26 0.18	0.20	0.07			0.08 0.05	0.07
FCR	LR♂	0.0152			0.0207 0.0224	0.0194	0.23			0.16 0.18	0.19	0.18			0.12 0.07	0.12
	LR♀	0.0233			0.0245 0.0285	0.0254	0.16			0.26 0.22	0.21	0.04			0.04 0.06	0.05
	LW♂	0.0186		0.00845	0.0183 0.0171	0.0156	0.20		0.32	0.22 0.23	0.24	0.15		0.08	0.09 0.11	0.11
	LW♀	0.0152			0.0190 0.0299	0.0214	0.30			0.37 0.20	0.29	0.07			0.05 0.04	0.05

* - subscript 1 refers to TMG codes for LR♂, LR♀ or LW♀; and 2 to LW♂; $\bar{\sigma}_e^2$, $\bar{h}^2$ and $\bar{c}^2$ are averages of residual variances, heritabilities and common litter of birth effects across TMGs.

Table 2.7: Averages of residual variance, heritability and common litter of birth effects within sex and Test Management Group and across breed for selected traits. Model of analysis included additive direct and common litter of birth random effects only; full results appear in tables 2.5.i - 2.5.xiii.

Trait:	Sex	σ_e^2			h^2			c^2		
		TMG:			TMG:			TMG:		
		1_1	2	3	1_1	2	3	1_1	2	3
C Fat	♂	1.45	1.15	1.43	0.32	0.48	0.35	0.10	0.02	0.01
	♀	1.42	1.31	1.13	0.45	0.35	0.42	0.04	0.07	0.03
ADFI	♂	0.0152	0.0175	0.0224	0.21	0.24	0.12	0.10	0.07	0.10
	♀	0.00487	0.0134	0.0113	0.11	0.16	0.06	0.09	0.09	0.08
ADG	♂	0.00544	0.00599	0.00799	0.28	0.22	0.19	0.06	0.02	0.01
	♀	0.00217	0.00412	0.00449	0.14	0.23	0.17	0.08	0.09	0.08
FCR	♂	0.0141	0.0195	0.0198	0.25	0.19	0.21	0.14	0.11	0.09
	♀	0.0193	0.0218	0.0292	0.23	0.32	0.21	0.06	0.05	0.05

1 - for boar TMG 1 average is of estimates from TMG1 of LR♂ and from TMGs 1 and 2 of LW♀.

Within a breed and sex combination, heritability estimates, although differing in magnitude between models, tended to show the same trend across test management groups for all models. That is, if the heritability estimate from model 1 decreased from the first test management group to the last, it was generally observed to do so for models 2 and 3 also. Therefore, the following description of the differences across Test Management Groups does not refer to a specific model.

In gilt data sets an increase in the estimate of heritability when the food allowance was increased, followed by a subsequent decrease when the health status of the herd declined, was observed for ADFI, WtG, ADG, and FCR in both Landrace and Large White breeds.

With regards to the trends across Test Management Groups for heritability estimates for the ultrasonic backfat depths, the results from gilt data sets differed between the breeds. In both breeds the heritability of L Fat declined across Test Management Groups. This trend was repeated for Sh Fat in the Landrace breed and C Fat and K Fat in the Large White breed. For C Fat and K Fat in the Landrace breed and Sh Fat in the Large White breed the heritability estimates decreased when the food allowance was increased, and then increased in the low health status test management group.

In boars, the heritability estimates for ADFI were increased in the Test Management Groups where feed was less restricted compared to the first test management group, and decreased when the health status of the herd declined. This was repeated for WtG, ADG and FCR in the Large White breed, while these growth traits in Landrace showed a steady decline across groups for FCR and a decrease for WtG and ADG in group 2

followed by an increase in the low health status group.

In general, heritability estimates were comparable across breeds until the major food allowance increase half way through the data collection period, which resulted in higher estimates in the following group for Large Whites than for Landrace.

Estimates of the litter of birth effect were fairly consistent in both magnitude and statistical significance across gilt test management groups for both breeds. In boar data sets there was a tendency towards lower estimates (with a corresponding decrease in the test statistic) in later test management groups.

Although the zero estimates of m^2 in tables 2.9.i-2.9.xiii were due to rounding, rather than true zero estimates, few estimates of the additive maternal effect and additive by additive covariance gave significant test statistics. In some cases where there were appreciable estimates of m^2 and σ_{am}/σ_p^2 for a given trait, it appeared that the two components acted to cancel each other out, giving a non-significant value for the test statistic, for example the results for ADG from Large White boars from months of test 19 to 35. The test statistic for the maternal effect was significant ($p<0.1$) in only six analyses, five of which were in the final test management groups for Large White boars and gilts, with the traits being WtG, ADG and FCR.

Table 2.8.i: Results of univariate DFREML analyses fitting additive direct, additive maternal, additive-additive covariance and cytoplasmic line random effects in Landrace boar data sets.

Trait	TMG	TS	σ_e^2	σ_p^2	h^2	c_{cyto}^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1	8.048	5.873	6.984	0.16	0.00	0.14	-0.07	-0.48
	2	0.046	5.569	7.525	0.30	0.00	0.00	-0.02	-0.93
	3	0.728	8.477	9.501	0.06	0.01	0.01	0.02	0.91
L Fat	1	0.766	1.593	2.062	0.22	0.00	0.03	-0.01	-0.13
	2	0.014	1.697	1.884	0.16	0.01	0.01	-0.04	-0.93
	3	-0.116	2.156	2.157	0.02	0.00	0.02	-0.02	-1.00
C Fat	1	2.350	1.394	2.115	0.28	0.00	0.01	0.03	0.49
	2	0.726	1.398	2.180	0.27	0.00	0.01	0.04	0.99
	3	3.040	1.258	1.866	0.54	0.00	0.05	-0.13	-0.79
K Fat	1	4.882	1.498	2.033	0.26	0.00	0.10	-0.05	-0.29
	2	0.264	1.360	2.349	0.35	0.00	0.01	0.04	0.82
	3	5.830	1.480	1.848	0.34	0.00	0.12	-0.13	-0.65
ADFI	1	6.926	0.015	0.020	0.19	0.00	0.01	-0.03	-0.20
	2	1.150	0.018	0.027	0.26	0.00	0.01	0.04	1.00
	3	1.976	0.025	0.030	0.08	0.01	0.02	0.04	0.99
WtG	1	0.856	15.187	19.466	0.22	0.01	0.01	-0.01	-0.14
	2	0.210	17.360	18.657	0.06	0.00	0.01	-0.00	-0.05
	3	1.768	21.651	24.417	0.15	0.02	0.07	-0.06	-0.62
ADG	1	0.838	0.006	0.008	0.22	0.01	0.01	-0.01	-0.21
	2	0.480	0.007	0.007	0.07	0.00	0.01	-0.00	-0.04
	3	1.818	0.008	0.009	0.15	0.01	0.08	-0.07	-0.62
FCR	1	0.268	0.016	0.026	0.40	0.01	0.01	-0.02	-0.50
	2	3.556	0.023	0.029	0.14	0.00	0.01	0.04	0.99
	3	0.090	0.023	0.030	0.21	0.00	0.01	0.00	0.06

TMG is test management group; TS = $-2(L_1-L_{cyto})$

Table 2.8.ii: Results of univariate DFREML analyses fitting additive direct, additive maternal, additive-additive covariance and cytoplasmic line random effects in Landrace gilt data sets.

Trait	TMG	TS	σ^2_e	σ^2_p	h^2	c^2_{cyto}	m^2	σ_{am}/σ^2_p	r_{am}
Sh Fat	1	1.212	5.950	8.920	0.39	0.00	0.01	-0.03	-0.46
	2	1.130	6.001	8.679	0.40	0.00	0.01	-0.05	-0.90
	3	2.910	6.769	8.080	0.10	0.00	0.01	0.03	0.77
L Fat	1	0.078	1.806	2.592	0.27	0.00	0.01	0.01	0.40
	2	4.972	1.732	2.033	0.09	0.00	0.01	0.02	0.99
	3	1.054	1.807	2.025	0.06	0.01	0.01	0.02	0.91
C Fat	1	1.736	1.293	2.363	0.52	0.01	0.04	-0.06	-0.39
	2	1.322	1.191	1.881	0.41	0.00	0.01	-0.03	-0.33
	3	1.008	1.025	1.831	0.43	0.00	0.02	-0.01	-0.07
K Fat	1	4.798	1.329	2.294	.54	0.01	0.09	-0.11	-0.50
	2	1.326	1.255	1.935	0.41	0.00	0.02	-0.04	-0.43
	3	1.376	1.105	1.822	0.41	0.00	0.03	-0.02	-0.19
ADFI	1	5.838	0.005	0.006	0.07	0.00	0.03	0.02	0.43
	2	4.948	0.013	0.017	0.18	0.00	0.01	0.04	1.00
	3	6.760	0.011	0.011	0.08	0.01	0.14	-0.10	-0.97
WtG	1	0.884	8.247	9.396	0.16	0.01	0.00	-0.02	-0.99
	2	1.924	10.619	14.046	0.17	0.00	0.01	0.03	0.98
	3	2.006	13.806	17.150	0.15	0.01	0.01	0.02	0.59
ADG	1	0.704	0.002	0.003	0.15	0.01	0.00	-0.02	-0.98
	2	1.980	0.004	0.006	0.18	0.00	0.01	0.03	0.95
	3	2.108	0.004	0.005	0.15	0.01	0.01	0.02	0.74
FCR	1	0.554	0.023	0.029	0.23	0.00	0.01	-0.03	-0.53
	2	0.680	0.023	0.034	0.38	0.00	0.01	-0.04	-0.57
	3	0.360	0.029	0.040	0.29	0.00	0.01	-0.01	-0.22

TMG is test management group; TS = $-2(L_1-L_{cyto})$

Table 2.9.i: Univariate REML analysis results - LR boar performance test data from Test Management Group 1.

Trait	m[a]	-2(L_{m-1}- L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		5.39	7.72	0.30 (0.05)				
	2	4.796	5.34	7.61	0.22 (0.05)	0.08 (0.03)			
	3	8.090	5.64	7.16	0.15	0.06	0.10	-0.05	-0.39
L Fat	1		1.56	2.10	0.26 (0.05)				
	2	0.976	1.54	2.09	0.23 (0.05)	0.04 (0.03)			
	3	0.128	1.52	2.10	0.21	0.05	0.02	0.00	0.00
C Fat	1		1.33	2.08	0.36 (0.07)				
	2	8.034	1.28	2.06	0.28 (0.06)	0.10 (0.03)			
	3	1.596	1.35	2.04	0.23	0.07	0.04	0.00	0.00
K Fat	1		1.39	2.18	0.36 (0.07)				
	2	4.410	1.36	2.16	0.30 (0.06)	0.07 (0.03)			
	3	1.974	1.45	2.13	0.22	0.05	0.05	0.00	0.00
ADFI	1		0.0148	0.0208	0.29 (0.07)				
	2	6.984	0.0143	0.0205	0.22 (0.06)	0.09 (0.03)			
	3	3.556	0.0148	0.0199	0.17	0.07	0.07	-0.02	-0.22
WtG	1		15.2	19.6	0.22 (0.06)				
	2	1.824	14.8	19.6	0.20 (0.06)	0.05 (0.03)			
	3	0.684	14.7	19.2	0.21	0.04	0.03	-0.02	-0.27
ADG	1		0.00595	0.00773	0.23 (0.06)				
	2	1.806	0.00582	0.00770	0.20 (0.06)	0.05 (0.03)			
	3	0.664	0.00580	0.00754	0.20	0.04	0.03	-0.02	-0.28
FCR	1		0.0168	0.0262	0.36 (0.08)				
	2	26.792	0.0152	0.0258	0.23 (0.06)	0.18 (0.03)			
	3	1.310	0.0139	0.0247	0.35	0.19	0.01	-0.06	-0.97

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.ii: Univariate REML analysis results - LR boar performance test data from Test Management Group 2.

Trait	m[a]	-2(L_{m-1} - L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		5.84	7.59	0.23 (0.09)				
	2	0.048	5.76	7.61	0.24 (0.09)	0.01 (0.06)			
	3	0.028	5.71	7.58	0.23	0.01	0.00	-0.00	-0.47
L Fat	1		1.72	1.94	0.12 (0.07)				
	2	0.000	1.71	1.95	0.12 (0.12)	0.00 (0.05)			
	3	0.030	1.71	1.94	0.12	0.00	0.00	-0.00	-1.00
C Fat	1		1.27	2.13	0.40 (0.12)				
	2	0.164	1.33	2.10	0.36 (0.10)	0.01 (0.05)			
	3	0.444	1.33	2.09	0.30	0.06	0.00	0.00	0.29
K Fat	1		1.30	2.28	0.43 (0.13)				
	2	1.812	1.35	2.22	0.31 (0.11)	0.08 (0.05)			
	3	0.012	1.34	2.23	0.31	0.08	0.00	0.00	0.26
ADFI	1		0.0168	0.0262	0.36 (0.14)				
	2	2.250	0.0178	0.0254	0.19 (0.12)	0.11 (0.06)			
	3	0.066	0.0178	0.0254	0.19	0.10	0.00	0.00	0.51
WtG	1		17.2	18.7	0.08 (0.08)				
	2	0.172	17.2	18.7	0.08 (0.14)	0.01 (0.06)			
	3	0.250	16.9	18.7	0.05	0.04	0.00	0.00	0.75
ADG	1		0.00665	0.00717	0.07 (0.07)				
	2	0.162	0.00657	0.00718	0.08 (0.14)	0.01 (0.06)			
	3	0.268	0.00651	0.00719	0.05	0.04	0.00	0.00	0.80
FCR	1		0.0213	0.0288	0.26 (0.12)				
	2	2.344	0.0207	0.0286	0.16 (0.12)	0.12 (0.06)			
	3	0.262	0.0205	0.0287	0.16	0.11	0.00	0.00	0.95

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.iii: Univariate REML analysis results - LR boar performance test data from Test Management Group 3.

Trait	m[a]	-2(L_{m-1} - L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		8.19	9.41	0.13 (0.10)				
	2	0.142	8.12	9.41	0.13 (0.11)	0.01 (0.04)			
	3	0.390	8.28	9.36	0.07	0.04	0.00	0.00	0.67
L Fat	1		2.20	2.20	0.00				
	2	0.068	2.18	2.20	0.00	0.01 (0.05)			
	3	-0.026	2.16	2.20	0.00	0.02	0.00	0.00	-0.87
C Fat	1		1.39	2.08	0.33 (0.11)				
	2	0.132	1.36	2.09	0.34 (0.11)	0.01 (0.06)			
	3	0.160	1.35	2.09	0.32	0.03	0.00	0.00	0.16
K Fat	1		1.49	2.13	0.30 (0.10)				
	2	0.026	1.48	2.13	0.30 (0.10)	0.01 (0.06)			
	3	0.900	1.35	2.08	0.42	0.01	0.00	-0.04	-1.00
ADFI	1		0.0239	0.0294	0.19 (0.10)				
	2	5.438	0.0226	0.0291	0.09 (0.07)	0.14 (0.05)			
	3	0.034	0.0226	0.0292	0.08	0.14	0.00	0.00	0.66
WtG	1		21.6	26.1	0.17 (0.08)				
	2	0.000	21.5	26.1	0.17 (0.08)	0.00 (0.02)			
	3	0.006	21.6	26.1	0.17	0.00	0.00	0.00	0.27
ADG	1		0.00831	0.01005	0.17 (0.08)				
	2	-0.002	0.00828	0.01006	0.17 (0.08)	0.00			
	3	0.030	0.00832	0.01007	0.17	0.00	0.00	0.00	0.36
FCR	1		0.0231	0.0301	0.23 (0.10)				
	2	1.910	0.0224	0.0299	0.18 (0.08)	0.07 (0.05)			
	3	0.139	0.0229	0.0298	0.18	0.08	0.00	-0.01	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.iv: Univariate REML analysis results - LR gilt performance test data from Test Management Group 1.

Trait	m[a]	-2(L_{m-1}- L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		6.24	9.13	0.32				
	2	1.114	6.18	9.12	0.30	0.02			
	3	0.808	6.02	9.01	0.34	0.02	0.00	-0.00	-1.00
L Fat	1		1.73	2.58	0.33				
	2	9.180	1.72	2.55	0.27	0.06			
	3	0.000	1.73	2.55	0.26	0.06	0.00	0.00	0.26
C Fat	1		1.36	2.48	0.45				
	2	18.728	1.33	2.44	0.39	0.07			
	3	1.082	1.24	2.38	0.47	0.08	0.00	-0.04	-0.87
K Fat	1		1.39	2.54	0.45				
	2	14.554	1.37	2.50	0.39	0.07			
	3	0.884	1.29	2.45	0.46	0.07	0.00	-0.03	-0.98
ADFI	1		0.00475	0.00564	0.16				
	2	14.828	0.00467	0.00558	0.09	0.07			
	3	1.530	0.00476	0.00560	0.06	0.06	0.02	0.01	0.22
WtG	1		8.29	9.61	0.14				
	2	14.388	7.94	9.59	0.10	0.07			
	3	0.024	7.90	9.59	0.11	0.07	0.00	-0.00	-0.14
ADG	1		0.00235	0.00272	0.14				
	2	17.138	0.00222	0.00272	0.10	0.08			
	3	0.280	0.00223	0.00271	0.11	0.08	0.00	-0.00	-0.63
FCR	1		0.0238	0.0292	0.19				
	2	5.884	0.0233	0.0292	0.16	0.04			
	3	0.030	0.0232	0.0291	0.16	0.05	0.00	-0.00	-0.10

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.v: Univariate REML analysis results - LR gilt performance test data from Test Management Group 2.

Trait	m[a]	-2(L_{m-1} - L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		6.48	8.93	0.27				
	2	6.016	6.47	8.83	0.21	0.06			
	3	1.016	6.37	8.75	0.24	0.05	0.00	-0.01	-0.99
L Fat	1		1.62	2.03	0.20				
	2	15.784	1.65	1.98	0.07	0.10			
	3	0.312	1.66	1.98	0.06	0.10	0.00	0.00	0.59
C Fat	1		1.21	1.93	0.37				
	2	13.000	1.25	1.88	0.25	0.09			
	3	0.770	1.21	1.86	0.30	0.09	0.00	-0.02	-1.00
K Fat	1		1.27	2.03	0.37				
	2	13.006	1.31	1.97	0.25	0.09			
	3	1.400	1.25	1.93	0.32	0.09	0.00	-0.03	-1.00
ADFI	1		0.0121	0.0170	0.29				
	2	26.528	0.0118	0.0167	0.17	0.12			
	3	0.104	0.0119	0.0166	0.15	0.12	0.00	0.00	0.27
WtG	1		10.0	13.8	0.27				
	2	12.988	9.9	13.6	0.19	0.08			
	3	0.080	9.8	13.7	0.19	0.08	0.00	0.00	0.33
ADG	1		0.00401	0.00551	0.27				
	2	13.048	0.00391	0.00545	0.20	0.09			
	3	0.082	0.00394	0.00546	0.19	0.08	0.00	0.00	0.33
FCR	1		0.0246	0.0349	0.30				
	2	2.476	0.0245	0.0347	0.26	0.04			
	3	0.926	0.0237	0.0341	0.31	0.04	0.00	-0.02	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.vi: Univariate REML analysis results - LR gilt performance test data from Test Management Group 3.

Trait	m[a]	-2(L_{m-1} - L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		6.32	8.06	0.22				
	2	9.346	6.44	7.86	0.11	0.07			
	3	0.228	6.46	7.87	0.10	0.07	0.00	0.00	0.42
L Fat	1		1.83	1.97	0.07				
	2	5.474	1.81	1.96	0.03	0.05			
	3	0.452	1.81	1.97	0.02	0.05	0.00	0.00	0.99
C Fat	1		0.92	1.91	0.52				
	2	1.658	0.97	1.87	0.46	0.02			
	3	0.022	0.98	1.86	0.44	0.02	0.00	0.00	0.32
K Fat	1		1.03	1.91	0.46				
	2	1.836	1.07	1.88	0.41	0.02			
	3	0.022	1.08	1.87	0.40	0.02	0.00	0.00	0.26
ADFI	1		0.0107	0.0129	0.17				
	2	25.082	0.0105	0.0127	0.05	0.12			
	3	0.236	0.0105	0.0126	0.05	0.12	0.00	-0.00	-1.00
WtG	1		13.2	17.1	0.23				
	2	17.556	12.9	16.9	0.15	0.09			
	3	0.052	12.8	16.8	0.15	0.09	0.00	-0.00	-0.98
ADG	1		0.00414	0.00538	0.23				
	2	18.620	0.00403	0.00531	0.15	0.10			
	3	0.048	0.00399	0.00529	0.16	0.10	0.00	-0.01	-1.00
FCR	1		0.0286	0.0402	0.29				
	2	9.010	0.0285	0.0396	0.22	0.06			
	3	0.800	0.0273	0.0389	0.28	0.07	0.00	-0.03	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.vii: Univariate REML analysis results - LW boar performance test data from Test Managment Group 1.

Trait	m[a]	-2(L_{m-1} - L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		7.43	11.61	0.36 (0.09)				
	2	2.956	7.48	11.47	0.27 (0.08)	0.07 (0.04)			
	3	1.590	7.23	9.74	0.37	0.08	0.17	-0.18	-0.73
L Fat	1		2.23	2.94	0.24 (0.07)				
	2	9.364	2.09	2.94	0.16 (0.06)	0.12 (0.04)			
	3	1.166	2.03	2.82	0.22	0.14	0.01	-0.05	-1.00
C Fat	1		1.51	2.59	0.41 (0.08)				
	2	4.422	1.49	2.57	0.34 (0.07)	0.08 (0.04)			
	3	0.298	1.40	2.48	0.42	0.10	0.01	-0.04	-0.73
K Fat	1		1.53	2.71	0.44 (0.08)				
	2	2.850	1.52	2.69	0.37 (0.08)	0.07 (0.04)			
	3	0.414	1.43	2.62	0.45	0.08	0.00	-0.04	-0.99
ADFI	1		0.0186	0.0225	0.17 (0.08)				
	2	4.776	0.0179	0.0225	0.10 (0.06)	0.10 (0.05)			
	3	1.234	0.0182	0.0229	0.07	0.07	0.03	0.02	0.44
WtG	1		15.9	22.6	0.30 (0.09)				
	2	2.840	15.7	22.5	0.23 (0.08)	0.07 (0.04)			
	3	0.600	16.2	23.1	0.18	0.06	0.01	0.03	0.99
ADG	1		0.00637	0.00905	0.30 (0.09)				
	2	2.856	0.00629	0.00900	0.23 (0.08)	0.07 (0.04)			
	3	0.458	0.00648	0.00913	0.18	0.06	0.02	0.02	0.32
FCR	1		0.0193	0.0292	0.34 (0.08)				
	2	15.318	0.0186	0.0287	0.20 (0.07)	0.15 (0.04)			
	3	0.246	0.0181	0.0279	0.24	0.17	0.01	-0.03	-0.80

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.viii: Univariate REML analysis results - LW boar performance test data from Test Management Group 2.

Trait	m[a]	$-2(L_{m-1} - L_m)$[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		8.32	10.53	0.21 (0.08)				
	2	6.958	7.81	10.49	0.13 (0.06)	0.13 (0.05)			
	3	0.146	7.90	10.61	0.11	0.12	0.00	0.01	0.99
L Fat	1		1.83	2.55	0.28 (0.08)				
	2	-0.002	1.82	2.55	0.29 (0.08)	0.00			
	3	0.226	1.79	2.51	0.33	0.00	0.00	-0.02	-1.00
C Fat	1		1.68	2.96	0.43 (0.10)				
	2	5.538	1.58	2.95	0.35 (0.09)	0.12 (0.05)			
	3	1.610	1.41	2.77	0.49	0.14	0.01	-0.08	-1.00
K Fat	1		1.73	2.91	0.41 (0.09)				
	2	3.520	1.64	2.91	0.34 (0.09)	0.09 (0.05)			
	3	1.222	1.53	2.80	0.43	0.10	0.01	-0.05	-1.00
ADFI	1		0.0144	0.0230	0.38 (0.10)				
	2	4.502	0.0135	0.0230	0.31 (0.09)	0.11 (0.05)			
	3	0.066	0.0139	0.0232	0.27	0.10	0.00	0.01	0.98
WtG	1		11.1	20.4	0.46 (0.10)				
	2	1.846	10.8	20.3	0.41 (0.10)	0.06 (0.05)			
	3	2.562	9.95	16.3	0.59	0.07	0.25	-0.26	-0.68
ADG	1		0.00432	0.00797	0.46 (0.10)				
	2	1.724	0.00420	0.00794	0.41 (0.10)	0.06 (0.05)			
	3	2.604	0.00390	0.00635	0.59	0.07	0.24	-0.26	-0.68
FCR	1		0.00857	0.0143	0.40 (0.09)				
	2	2.902	0.00845	0.0141	0.32 (0.08)	0.08 (0.05)			
	3	1.780	0.00774	0.0129	0.46	0.13	0.02	-0.10	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.ix: Univariate REML analysis results - LW boar performance test data from Test Management Group 3.

Trait	m[a]	$-2(L_{m-1} - L_m)^b$	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		5.58	12.13	0.54 (0.09)				
	2	3.062	5.79	11.85	0.45 (0.09)	0.06 (0.03)			
	3	1.836	7.03	12.20	0.23	0.05	0.02	0.06	1.00
L Fat	1		1.80	2.41	0.26 (0.07)				
	2	0.000	1.80	2.41	0.25 (0.07)	0.00 (0.00)			
	3	0.542	1.86	2.45	0.19	0.00	0.00	0.02	1.00
C Fat	1		0.969	2.49	0.61 (0.09)				
	2	0.028	0.978	2.48	0.60 (0.09)	0.01 (0.03)			
	3	0.030	0.950	2.45	0.64	0.00	0.00	-0.01	-1.00
K Fat	1		1.24	2.57	0.52 (0.09)				
	2	0.000	1.24	2.57	0.52 (0.09)	0.00			
	3	0.006	1.22	2.57	0.54	0.00	0.00	-0.00	-0.74
ADFI	1		0.0172	0.0252	0.32 (0.08)				
	2	0.354	0.0172	0.0250	0.29 (0.08)	0.02 (0.03)			
	3	1.100	0.0183	0.0253	0.20	0.02	0.02	0.02	0.33
WtG	1		14.0	23.3	0.40 (0.08)				
	2	0.898	14.0	23.2	0.36 (0.08)	0.03 (0.03)			
	3	0.512	14.8	23.7	0.28	0.03	0.00	0.03	1.00
ADG	1		0.00542	0.00900	0.40 (0.08)				
	2	0.870	0.00541	0.00894	0.36 (0.08)	0.03 (0.03)			
	3	0.486	0.00571	0.00914	0.28	0.03	0.00	0.03	1.00
FCR	1		0.0180	0.0271	0.34 (0.09)				
	2	6.902	0.0183	0.0264	0.22 (0.07)	0.09 (0.03)			
	3	0.326	0.0188	0.0266	0.18	0.08	0.02	0.01	0.20

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.x: Univariate REML analysis results - LW boar performance test data from Test Management Group 4.

Trait	m[a]	$-2(L_{m-1} - L_m)$[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		8.28	10.15	0.18 (0.07)				
	2	0.000	8.29	10.15	0.18 (0.07)	0.00			
	3	3.546	7.38	8.86	0.42	0.00	0.09	-0.17	-0.87
L Fat	1		1.93	2.20	0.12 (0.07)				
	2	3.000	1.88	2.18	0.07 (0.06)	0.07 (0.04)			
	3	1.814	1.85	2.03	0.11	0.08	0.05	-0.07	-1.00
C Fat	1		1.49	2.32	0.36 (0.09)				
	2	0.000	1.49	2.32	0.36 (0.09)	0.00			
	3	3.810	1.47	2.05	0.41	0.00	0.13	-0.13	-0.55
K Fat	1		1.52	2.46	0.38 (0.10)				
	2	0.850	1.54	2.43	0.33 (0.09)	0.04 (0.04)			
	3	2.824	1.54	2.18	0.37	0.02	0.13	-0.11	-0.51
ADFI	1		0.0221	0.0274	0.19 (0.09)				
	2	1.206	0.0222	0.0271	0.14 (0.07)	0.05 (0.04)			
	3	1.812	0.0230	0.0280	0.08	0.02	0.02	0.03	1.00
WtG	1		20.0	25.7	0.22 (0.08)				
	2	0.050	20.0	25.7	0.21 (0.08)	0.01 (0.04)			
	3	4.606	20.0	23.4	0.22	0.00	0.09	-0.09	-0.59
ADG	1		0.00771	0.00990	0.22 (0.08)				
	2	0.058	0.00769	0.00988	0.21 (0.08)	0.01 (0.05)			
	3	5.038	0.00741	0.00853	0.29	0.00	0.16	-0.16	-0.76
FCR	1		0.0163	0.0278	0.39 (0.11)				
	2	5.100	0.0171	0.0258	0.23 (0.09)	0.11 (0.04)			
	3	5.398	0.0170	0.0198	0.31	0.13	0.29	-0.30	-0.97

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.xi: Univariate REML analysis results - LW gilt performance test data from Test Management Group 1.

Trait	m[a]	-2(L_{m-1}- L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		7.04	10.14	0.31 (0.05)				
	2	4.523	6.94	10.08	0.27 (0.04)	0.04 (0.02)			
	3	0.534	6.75	9.93	0.31	0.04	0.001	-0.02	-1.00
L Fat	1		1.98	2.84	0.30 (0.04)				
	2	4.471	1.95	2.82	0.27 (0.04)	0.04 (0.02)			
	3	2.257	1.87	2.78	0.33	0.04	0.002	-0.02	-1.00
C Fat	1		1.52	3.08	0.51 (0.05)				
	2	0.340	1.51	3.07	0.50 (0.05)	0.01 (0.02)			
	3	2.719	1.51	2.95	0.52	0.002	0.039	-0.04	-0.25
K Fat	1		1.54	3.22	0.52 (0.05)				
	2	0.288	1.53	3.21	0.51 (0.05)	0.01 (0.02)			
	3	1.093	1.38	3.20	0.60	0.01	0.011	-0.03	-0.34
ADFI	1		0.00528	0.00668	0.21 (0.04)				
	2	30.904	0.00507	0.00659	0.12 (0.03)	0.11 (0.02)			
	3	0.292	0.00504	0.00655	0.13	0.11	0.000	-0.01	-1.00
WtG	1		7.61	9.80	0.22 (0.04)				
	2	12.203	7.41	9.71	0.17 (0.04)	0.07 (0.02)			
	3	1.653	7.23	9.62	0.22	0.06	0.001	-0.02	-0.99
ADG	1		0.00217	0.00281	0.23 (0.04)				
	2	13.820	0.00211	0.00279	0.17 (0.04)	0.07 (0.02)			
	3	1.458	0.00209	0.00276	0.19	0.08	0.001	-0.01	-1.00
FCR	1		0.0157	0.0246	0.36 (0.05)				
	2	15.756	0.0152	0.0243	0.30 (0.05)	0.07 (0.02)			
	3	4.544	0.0143	0.0233	0.40	0.08	0.006	-0.05	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.xii: Univariate REML analysis results - LW gilt performance test data from Test Management Group 2.

Trait	m[a]	-2(L_{m-1}- L_m)[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		8.11	12.30	0.34 (0.07)				
	2	8.104	8.57	11.91	0.21 (0.06)	0.07 (0.02)			
	3	2.054	9.03	11.67	0.13	0.06	0.06	-0.01	-0.12
L Fat	1		1.92	2.55	0.25 (0.06)				
	2	1.226	1.92	2.53	0.22 (0.05)	0.02 (0.02)			
	3	3.760	1.94	2.21	0.23	0.02	0.14	-0.13	-0.76
C Fat	1		1.28	2.70	0.53 (0.07)				
	2	4.234	1.36	2.63	0.44 (0.07)	0.05 (0.02)			
	3	0.060	1.31	2.61	0.48	0.05	0.00	-0.01	-0.95
K Fat	1		1.25	2.74	0.55 (0.07)				
	2	9.484	1.35	2.64	0.42 (0.07)	0.07 (0.02)			
	3	0.456	1.25	2.58	0.51	0.08	0.00	-0.04	-1.00
ADFI	1		0.0149	0.0189	0.21 (0.06)				
	2	5.806	0.0149	0.0186	0.14 (0.05)	0.06 (0.02)			
	3	0.060	0.0150	0.0187	0.13	0.06	0.00	0.00	0.99
WtG	1		10.6	16.2	0.35 (0.06)				
	2	12.352	10.6	16.0	0.26 (0.05)	0.08 (0.02)			
	3	0.366	10.5	15.9	0.27	0.08	0.00	-0.01	-0.99
ADG	1		0.00435	0.00658	0.34 (0.06)				
	2	11.854	0.00433	0.00650	0.26 (0.05)	0.08 (0.02)			
	3	0.472	0.00430	0.00645	0.26	0.08	0.00	-0.01	-1.00
FCR	1		0.0185	0.0338	0.45 (0.06)				
	2	6.886	0.0190	0.0332	0.37 (0.06)	0.05 (0.02)			
	3	0.476	0.0182	0.0330	0.42	0.05	0.00	-0.01	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

Table 2.9.xiii: Univariate REML analysis results - LW gilt performance test data from Test Management Group 3.

Trait	m[a]	$-2(L_{m-1}- L_m)$[b]	σ_e^2	σ_p^2	h^2	c^2	m^2	σ_{am}/σ_p^2	r_{am}
Sh Fat	1		7.73	11.37	0.32 (0.06)				
	2	4.474	7.83	11.19	0.26 (0.06)	0.04 (0.02)			
	3	1.004	8.08	11.00	0.21	0.03	0.04	-0.01	-0.12
L Fat	1		1.93	2.31	0.17 (0.05)				
	2	4.070	1.92	2.29	0.12 (0.04)	0.04 (0.02)			
	3	0.356	1.94	2.27	0.11	0.04	0.02	-0.01	-0.16
C Fat	1		1.28	2.17	0.41 (0.06)				
	2	3.208	1.29	2.15	0.37 (0.06)	0.03 (0.02)			
	3	0.432	1.32	2.13	0.34	0.03	0.02	-0.00	-0.05
K Fat	1		1.36	2.28	0.40 (0.06)				
	2	3.168	1.37	2.25	0.36 (0.06)	0.03 (0.02)			
	3	0.890	1.37	2.20	0.36	0.03	0.02	-0.02	-0.23
ADFI	1		0.0123	0.0135	0.09 (0.03)				
	2	5.528	0.0120	0.0136	0.07 (0.03)	0.04 (0.02)			
	3	1.914	0.0120	0.0133	0.08	0.05	0.00	-0.02	-1.00
WtG	1		15.8	20.0	0.21 (0.04)				
	2	8.042	15.3	20.0	0.19 (0.04)	0.05 (0.02)			
	3	7.588	14.7	19.1	0.26	0.06	0.01	-0.05	-1.00
ADG	1		0.00514	0.00645	0.20 (0.04)				
	2	8.616	0.00494	0.00646	0.18 (0.04)	0.05 (0.02)			
	3	10.748	0.00438	0.00579	0.40	0.07	0.05	-0.14	-1.00
FCR	1		0.0305	0.0395	0.23 (0.05)				
	2	5.470	0.0299	0.0394	0.20 (0.04)	0.04 (0.02)			
	3	6.472	0.0291	0.0379	0.25	0.05	0.01	-0.04	-1.00

[a] m = model as defined in text and table 2.2; [b] L_i is the estimate of the maximum log likelihood for model i; zero estimates are due to rounding.

DISCUSSION

The range of heritability estimates for ADG and backfat across all breed / sex / TMG / model combinations; 0.05 to 0.59 and 0.00 to 0.64, respectively; were comparable with those of recently published results; 0.11 to 0.52 and 0.28 to 0.63, respectively (see table 1.1). However, many of the extreme estimates were from analyses with the additive maternal effect and its covariance with the additive direct effect included. If the genetic covariance estimate was negative, the phenotypic variance estimate was decreased and the heritability estimate inflated. An example of this is the heritability estimate for ADG in LW boars from TMG 3. When the additive maternal effect is included; $h^2 = 0.59$, $\sigma_p^2 = 0.00635$ and $\sigma_{am}/\sigma_p^2 = -0.26$; but when just the additive direct and common litter of birth effects are fitted; $h^2 = 0.41$, $\sigma_p^2 = 0.00794$.

If we consider analyses under model 2 (common litter of birth effect included, but no additive maternal effect) then heritability estimates range from 0.08 to 0.41 for ADG and from 0.00 to 0.60 for backfat across breeds, sexes and Test Management Groups. Comparing these heritabilities with the recently published estimates, then most of the the new estimates are at the low end of the range. This may be because of differences in the methods of analysis and models fitted or because of population specific factors such as management, housing or feeding.

Differences across Test Management Groups.

Heritability estimates will be affected by the magnitude of the additive genetic and phenotypic variances. The phenotypic variance will be affected by the magnitude of its constituent (co)variances, which will change across

Test Management Groups within a breed and sex because of selection, the degree of restriction, the herds health status and (in the Landrace breed) the halothane status.

Selection:

Falconer (1981) shows two ways in which selection acts to reduce the additive genetic variance, by the generation of gametic phase disequilibrium, which reduces the between family variation, and the gradual fixing of alleles. Linkage disequilibrium would have had most of its effect after two generations of selection, whilst the fixing of alleles results in a gradual reduction over a period of time.

The estimates of heritability generated by the analyses are projections back to the base generation which is assumed to be unselected. This is not the case and therefore it is expected that the effect of linkage disequilibrium will have accrued before the data collection period began. Many of the traits across both breeds and sexes show a decrease in base population heritability estimates between the first and last Test Management Groups, however it is not possible to disassociate the gradual effect of selection from the more immediate, large magnitude effects of health status and degree of restriction. The estimates of heritability for the ultrasonic fat depths, in particular L Fat, show the most consistent downward trend across Test Management Groups, which will be due in part to selection on those traits.

Degree of restriction of expression:

Increasing the food allowance to each pig will allow greater expression of the pig's additive genetic component for appetite, but may also cause an increase in the residual variance for some traits due to there

being more room for recording errors. Within a level of restriction, environmental factors, such as temperature and health status, will also affect the amount eaten.

The effect of changing the degree of restriction would be expected to be most noticeable in estimates from gilt Test Management Groups, as gilts from the earliest TMG were the most restricted group of animals, and in both breeds an increase in the observed heritability of ADFI, WtG, ADG and FCR was found. These increases were also seen in Large White boars, while Landrace boar estimates of heritability for these traits decreased from TMG 1 to TMG 2. However, there was no indication in Figures 2.1 and 2.3 that Large White boars underwent a greater change in the degree of restriction between TMGs 1 and 2 than their Landrace contemporaries.

Health status:

In a high health status herd, we can assume that each pig's unique environmental component for disease will be equal. However once the herd's health status starts to decline this portion of the residual component will begin to vary between pigs, depending on how badly each pig is affected by the disease before and during the performance test. This will cause an increase in the residual variance in the population. This effect can be observed across both breeds and sexes for most traits, heritability estimates in the final Test Management Groups being lower than those in the preceeding group.

Halothane status:

For the Landrace breed the Test Management Groups differ with regard to the halothane status of pigs, a halothane testing system having been used to reduce the frequency of halothane sensitive pigs from an estimated 40% to almost zero during the data collection period. Halothane reactive pigs grow faster and are leaner than halothane non-reactors. The effect of decreasing the number of halothane sensitive pigs would be to remove the variation in growth and carcass traits caused by this gene, because an increasing proportion of the population have the same genotype for the halothane locus. Halothane sensitivity of pigs was not included as a factor in the analysis because not all pigs were tested.

Again, the changes in heritability caused by selection against halothane sensitivity will be confounded with other factors. However, if estimates from Landrace and Large Whites are compared and we assume that the only factor which will cause them to differ is halothane status, then it is expected that ultrasonic fat depths and growth rate traits would be more heritable in the first Landrace Test Management Groups than contemporary Large White groups. This is not observed. Following on from this, if we consider L Fat, which had similar heritability estimates in the first Test Management Groups of Landrace and Large Whites of both sexes, then differences observed between them in the final TMG may be due in part to the change in halothane status of the Landrace breed. These differences can be seen in the data, for model 1 the heritability of L Fat is 0.00, 0.12, 0.07 and 0.17 in the final Test Management Groups of LR♂, LW♂, LR♀ and LW♀.

Detection of additional random effects.

Common litter of birth effects:

From the results, it appears that there is conflicting evidence from boars and gilts regarding the presence of a common litter of birth effect in the data from both breeds, in that estimates of c^2 are consistently significant (probability of a greater Chi-squared value <0.1) across Test Management Groups, whilst estimates from boars in the first half of the data collection period were found to be more likely to be significant than those following the large food allowance increase and health status breakdown.

There are two possible explanations for this: either there is no litter of birth effect in the second half of the boar data, or there is some reason for failing to detect it.

If there is no common litter of birth effect in boar TMGs 2 and 3, this may indicate the presence of a common litter of birth effect only when feed is restricted by more than it was in the last two boar Test Management Groups. This could come about if the common litter of birth effects were present at the start of test and in less restricted test regimes were then overcome by compensatory feeding and growth.

In the following discussion it is assumed that the common litter of birth effect is present in all data sets (effectively giving more weight to the results from gilt data) and that there must, therefore, be an explanation as to why there are certain data sets from which the effect could not be detected.

The structure of the gilt performance test data across all test management groups is better suited to detection of common litter of birth effects than that of the boar data because there are more records per litter

on average and a lower percentage of litters represented by a single record (see table 2.1) which allows better separation of common litter of birth variation from residual variation.

Within the boar data, the estimation of c^2 in the first half of the data collection period gave statistically significant results. However, the data structure in boars is comparable across all Test Management Groups, with the average number of records of litter and percentage of litters represented by a single record being consistent. This implies that there must be some other factor affecting the c^2 estimation, which is having a greater effect upon boar estimates than gilt estimates. The two Test Management Groups which make up the second half of the data collection period were characterised by having an increased food allowance and a deteriorating health status respectively. It was expected, and observed, that these factors would have an effect upon the residual variation found for the traits and it may be this, coupled with the poorer data structure found in the boar data sets, that caused c^2 estimates in the second half of the data collection period to be lower in boars than gilts. This is because it is not possible for the program to partition the residual and common litter of birth effects correctly when there is only 1 (or very few) records per litter, and the increased residual variation in these Test Management Groups is swamping the system, by increasing the phenotypic variance and decreasing the magnitude of the c^2 effect.

Additive maternal genetic effects:

Out of the 104 analyses carried out (8 traits in 13 data sets/test management groups), only 6 detected an additive maternal genetic effect resulting in a likelihood ratio test statistic significant at the 10% probability level. Given that detection of common litter of birth effects was possible with

the data structure present in most Test Management Groups, it was expected that it would also be possible to estimate maternal effects since the percentage of dams represented by only one offspring and the average number of records per dam are more favourable than the corresponding values for litters.

The significant results were found in three Test Management Groups - LR boar Test Management Group 1, LW boar group 4 and LW gilt group 3, which represent low feeding and high health status in the LR group and high feeding with declining health status in the LW groups. There appears to be an additive maternal genetic effect for FCR, WtG and ADG in Large White pigs when the health status was declining. The estimates of the additive maternal effect for some traits in the final TMG could imply an additive genetic effect for resistance, or partial resistance, to the pathogen in the dam, with some degree of passive immunity in her offspring due to the passage of some factor, maybe an antibody, to the offspring via the dam's milk or blood supplies. However, this hypothesis would not explain the negative correlations between additive direct and additive maternal genetic effects found in these analyses.

From the parameters estimated under the animal model, it is possible to estimate genetic covariances between relatives. Generally, the genetic covariance between related individuals for a trait made up of genetic contributions for the trait itself and a maternal trait is (Willham, 1963):

$$cov(A_x,A_y) = 2p_{xy}\sigma_a^2 + [2p_{xz} + 2p_{wy}]\,\sigma_{am} + 2p_{wz}\sigma_m^2$$

Where: A_i - is the additive genetic value for individual i;

$2p_{ij}$ - Wright's coefficient of relationship for individuals i and j with no inbreeding;

x,y - related individuals of interest;

w,z - dams of x and y respectively.

Considering only additive genetic effects, the above formula can be summarised for various relationships in terms of the coefficients linked to the (co)variance components, as by Willham (1963) and Thompson (1976a):

Relationship:	σ_a^2	σ_{am}	σ_m^2
Paternal HS (PHS)	$\frac{1}{4}$	0	0
Maternal HS (MHS)	$\frac{1}{4}$	1	1
Full-sibs (FS_G)	$\frac{1}{2}$	1	1
Dam-Offspring (DO)	$\frac{1}{2}$	$\frac{5}{4}$	$\frac{1}{2}$
Sire-Offspring (SO)	$\frac{1}{2}$	$\frac{1}{4}$	0

Estimates of these between relative covariances and the phenotypic covariance between full-sibs ($FS_P = FS_G + c_c^2$ {Falconer, 1981}) can be produced, and these are given in table 2.10 using results from analyses which gave significant likelihood ratio statistics for the additive maternal genetic effect and the additive-additive covariance. For most of the other analyses performed, the contribution of the maternal genetic variance and the additive direct - additive maternal covariance to the phenotypic variance, and hence to estimates of the between relative covariances, was negligible.

Table 2.10: Estimates of between relative covariances for trait and Test Management Group combinations which resulted in significant likelihood ratio test statistics for a correlated additive maternal genetic effect.

Trait	Anal.[1]	Parameter estimate:				Between relative covariance:[2]					
		h^2	c^2	m^2	σ_{am}/σ_p^2	PHS	MHS	FS_G	FS_P	DO	SO
Sh Fat	LR♂1	0.23				0.06	0.06	0.11	0.11	0.11	0.11
		0.24	0.01			0.06	0.06	0.12	0.13	0.12	0.12
		0.15	0.06	0.10	-0.05	0.04	0.09	0.12	0.19	0.06	0.06
WtG	LW♀3	0.21				0.05	0.05	0.11	0.11	0.11	0.11
		0.19	0.05			0.05	0.05	0.09	0.14	0.09	0.09
		0.26	0.06	0.01	-0.05	0.06	0.02	0.09	0.15	0.07	0.12
ADG	LW♂4	0.22				0.05	0.05	0.11	0.11	0.11	0.11
		0.21	0.01			0.05	0.05	0.11	0.11	0.11	0.11
		0.29	0.00	0.16	-0.16	0.07	0.07	0.15	0.15	0.03	0.11
	LW♀3	0.20				0.05	0.05	0.10	0.10	0.10	0.10
		0.18	0.05			0.05	0.05	0.09	0.14	0.09	0.09
		0.40	0.07	0.05	-0.14	0.10	0.01	0.11	0.18	0.05	0.16
FCR	LW♂4	0.39				0.10	0.10	0.19	0.19	0.19	0.19
		0.23	0.11			0.06	0.06	0.11	0.22	0.11	0.11
		0.31	0.13	0.29	-0.30	0.08	0.07	0.15	0.27	-0.08	0.08
	LW♀3	0.23				0.06	0.06	0.11	0.11	0.11	0.11
		0.20	0.04			0.05	0.05	0.10	0.14	0.10	0.10
		0.25	0.05	0.01	-0.04	0.06	0.03	0.09	0.14	0.08	0.11

1 - Breed, Sex, Test Management Group (as defined in Table 2.1) combination;

2 - Expressed as a proportion of the phenotypic variance.

Large negative covariances between additive direct and additive maternal effects cause a decrease in the estimates of the dam-offspring and sire-offspring covariances. In the estimates of MHS and FS_G covariances the maternal genetic variance acts to counterbalance the effect of the negative additive direct-additive maternal genetic covariance, so that these estimates are stable across random effect models unless there was a large difference in the estimates of h^2 or m^2.

Dam-offspring covariance estimates show the most dramatic decline when a negatively correlated maternal effect was detected in the data, there being a lower coefficient attached to the additive maternal variance than to the additive direct-additive maternal covariance and hence less of a balancing out of effects as observed in MHS and FS_G covariance estimates.

Estimates of the correlation between additive genetic effects (direct and maternal) vary dramatically across analyses, with a tendency to be approximately ±1. Since most estimates of the additive maternal variance and additive direct - additive maternal covariance did not give statistically significant results, the high correlations between additive direct and maternal effects are of little concern.

Cytoplasmic line effects:

Although the data from the Landrace breed produced very few likelihood ratio test statistics which were significant at the 10% level, it is not possible to conclude that no cytoplasmic line effect exists. The model fitted assumed that there was a constant effect associated with each line, which implies no mutation of the cytoplasmic DNA and a constant copy number inherited by each animal from its dam (if expression, and subsequently the

size of the effect, is related to copy number), which are unlikely to be true, though the mutation rate may be negligible. Also it was assumed that base females were unique cytoplasmic sources and this assumption was known to be wrong when the analysis was begun, because the population had been a closed line undergoing selection for some time before the data collection period started and hence the dams were very likely to be related to one another. The consequence of this would be the presence of multiple lines with the same effect, resulting in a decrease in the variance between lines and increased difficulty of estimation. The result of breaking these assumptions would be to decrease the chance of finding a cytoplasmic effect which had a significant effect on the traits, and so although an effect was not found, it is not possible to conclude that one was not present.

SUMMARY AND CONCLUSIONS

Univariate estimates of heritabilities, common litter of birth effects, additive maternal genetic effects and cytoplasmic genetic effects were produced for performance test traits of Landrace and Large White boars and gilts. The data was analysed in groups corresponding to periods when different test regimes were in operation.

Heritability estimates from the analysis including the common litter of birth effect tended to be towards the low end of the range of recently published estimates. This may reflect either population specific effects, such as management, or the use of an Individual Animal Model.

Changes in heritability estimates across Test Management Groups were observed which corresponded to changes in health status, degree of restriction and halothane status. Changes due to selection were

confounded with these effects.

Estimates of the common litter of birth effect were around 0.05, and generally had a significant effect upon the fit of the model, while maternal and cytoplasmic effect estimates were negligible. Therefore, it is expected that omission of maternal and cytoplasmic effects from models for BLUP evaluation will not hinder genetic progress. Inclusion of common litter of birth effects would be recommended, in particular when the population of interest is fed on a restricted scale.

CHAPTER 3.

THE GENETIC RELATIONSHIP BETWEEN SEXES FOR PERFORMANCE TRAITS OF LANDRACE AND LARGE WHITE PIGS AS RECORDED IN A COMMERCIAL NUCLEUS HERD.

INTRODUCTION

It is common for pig breeders to select male and female pigs for the nucleus herd based upon the results of a performance test. However, with limited resources available for performance testing (both in terms of cash and test spaces) and still wishing to maximise genetic progress in the population, it makes sense to place greater emphasis on ensuring correct selection of boars, each of which is expected to have a greater genetic contribution to the coming generations than would be expected from a sow. Subsequently, boar testing tends to be more rigorous than gilt testing (often with individual housing and feeding in boars and group testing in gilts).

Feeding levels required in males and females are also assumed to be different, the assumption being made that gilts grow slower and do not therefore need the same amount of food to be offered to them. However, observation of the phenotypic variance for average daily food intake over a given time period (see figures 2.1 - 2.4; Chapter 2) indicates that the gilts are more restricted than their male contemporaries in this situation, which directly contradicts this assumption.

As a result of these practices it may be expected that there will be some degree of difference in the genetic control of a trait as measured in one sex and its analogue in the other sex. The aim of this study was to

produce estimates of the genetic correlations between the sexes for performance test traits, and use these as an indicator of the existence, or non-existence, of a genotype-environment interaction between boars and gilts undergoing different performance tests within the same pure breeding nucleus herd. This method was suggested by Falconer (1952), on the basis that if traits are genetically identical in both environments (in this case sexes) then the ranking of animals on their genotype will not change in either environment, and hence the genetic correlation will be equal to one. Although there can be no animal with a record on both male and female traits, the correlation is estimated using information on relatives.

Few estimates of genetic correlations between the sexes for performance test traits exist, Merks (1986) gave the most recent, making use of the covariance between male and female litter mates to estimate the correlation using Least-Squares to fit a sire model. Merks reported no evidence of genotype-sex interaction for the traits studied.

Earlier studies contain contradictory evidence of genotype-sex interactions. Roberts and Curran (1981) reported low genetic correlations between test performance of males and females, but their comparisons were made across very different test regimes with estimates of the genotype-sex interaction coming from farm tested males and station tested females and the reciprocal comparison. Smith and Ross (1965) analysed 26 traits recorded in a test station environment and found 9 estimates of sire-sex interactions which differed significantly from one. These nine included average daily gain and average backfat depth. Ollivier (1983) reported no evidence of genotype-sex interactions from data collected for a selection program, one with males and females in uniform environments.

There are many reports of the existence of genotype-environment interactions in pig breeding, for reviews of these results see Brascamp *et al.* (1985) and Webb and Curran (1986). However, Van Diepen and Kennedy (1990) applied a sophisticated mixed model approach to this problem and produced very high estimates of genetic correlations (and hence implied no GxE interaction) and from this de Vries and Sorensen (1990) raised the possibility that the estimates of GxE interaction previously quoted could be artefacts of the analysis used. This would also bring the previous estimates of genotype-sex interaction into question.

MATERIAL AND METHODS

Performance test records from male and female pigs of the Landrace and Large White breeds were available from a pure breeding nucleus herd. Selection within the herd was carried out using an index of traits recorded as described in Chapter 2 within each sex.

The principles of the test were similar in both males and females, in that growth rates (total weight gain on test {abbreviated to WtG throughout this text; recorded in kg} and average daily gain {ADG; kg}), average daily food intake (ADFI; kg), food conversion ratio (FCR; kg ADFI/kg ADG) and ultrasonic backfat depths (shoulder fat {Sh Fat; mm}, loin fat {L Fat; mm}, and approximate P2 and P3 fat depths {C Fat and K Fat; both mm}) were measured over the same range of weight (approximately 40-90 kg). Gilts were housed in groups of ten, however, and had a greater degree of restriction in the amount of food offered than boars, which were housed in pairs in a more controlled environment.

During the data collection period the nature of the test changed

somewhat. These changes were detected using data on ADFI and number of days spent on test, and to account for them, the data were divided into Test Management Groups, within breed and sex, as described in Chapter 2. Data from contemporary male and female Test Management Groups were then analysed together to estimate the between sex genetic correlations for the performance test traits, from which the presence or absence of a genotype-environment interaction can be inferred. In the Large White breed this resulted in the splitting of the first female test management group into two, to match up to the additional test management group found in the males. The population structure is summarised in table 3.1.

The data were analysed by fitting an individual animal model including random additive genetic and common litter of birth effects. No additive maternal effect was included, in accordance with the results found in Chapter 2.

Two assumptions were necessary for this analysis to be carried out; that there was a perfect correlation between common litter of birth effects between the sexes, which implies that the effect of being born in a given litter is the same on males and females, and that there was no within family environmental covariance between the sexes, that is, males and females are always tested separately and assigned to test groups (pairs for boars, groups of ten for gilts) at random.

Table 3.1: Summary of population structure within each data set analysed, and across the full data collection period within breed. Column headings as for table 2.1.

Breed	TMG	Sex	Period	n_{rec}	n_a	n_m	n_c	$c_{1\%}$	n_s	n_d	$d_{1\%}$
LR	1	♂	1-42	1607	4738	81	1113	14	68	477	7
		♀	1-39	2798							
	2	♂	43-57	538	2698	33	466	6	36	259	4
		♀	40-57	1743							
	3	♂	58-76	621	2709	38	445	7	28	241	6
		♀	58-76	1654							
	1-3	♂,♀	1-76	8961	9342	76	1937	9	99	752	4
LW	1	♂	1-18	873	2148	36	485	16	37	281	14
		♀	1-18	1016							
	2	♂	19-35	780	2824	38	608	15	49	335	7
		♀	19-39	1674							
	3	♂	36-57	1112	3328	38	584	15	53	312	13
		♀	40-55	1702							
	4	♂	58-77	884	3524	44	593	9	45	328	9
		♀	56-79	2100							
	1-4	♂,♀	1-79	10141	10545	79	2123	10	131	907	9

Different fixed effect structures were allowed for each sex, such that the effect of being tested in a given test month need not be the same on boars and gilts, and similarly for the covariates fitted. Within each sex the model fitted was:

$$Y_{ijklmn} = a_i + c_j + M_k + b1.L_{ijkl} + b2.W_{ijkm} + b3.A_{ijkn} + e_{ijklmn}$$

Where:

Y_{ijklmn} - is the phenotypic record of individual i for the trait;

a_i - is the additive genetic effect of the i^{th} individual;

c_j - is the random effect of the j^{th} litter (i.e. the litter into which animal i is born), giving rise to the common family environmental variance;

M_k - is the fixed effect associated with the k^{th} month of test;

$b1.L_{ijkl}$ - is the partial regression of Y on length of test period (days);

$b2.W_{ijkm}$ - is the partial regression of Y on weight at start of test (kg);

$b3.A_{ijkn}$ - is the partial regression of Y on age at start of test (days);

e_{ijklmn} - is the random environmental effect associated with the record, Y_{ijklmn}

The expectations of the additive genetic (a_{is}), common litter of birth (c_j) and residual random effects ($e_{ijklmns}$) were zero, and their variances were σ^2_{as}, σ^2_c and σ^2_{es} respectively, within sex s.

The analysis was performed using the univariate DFREML algorithm of Meyer (1989a), modified to perform bivariate analysis by a method proposed by Juga and Thompson (1990). This procedure makes use of the capacity of Meyer's algorithm to estimate two additive genetic effects (e.g. additive direct and additive maternal as used in Chapter 2). The analysis of two traits with records on an animal for a single trait only (as in this case with male and female records) and with different fixed effects associated with each trait was performed by expanding the vectors for observations and fixed effects, and the various incidence matrices as follows:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e} \qquad (1)$$

Equation (1) is a matrix representation of a general linear model where:

Y - is a vector of observations;

b - is a vector of fixed effects;

u - is a vector of random effects;

e - is a vector of random residual errors;

X,Z - are incidence matrices relating fixed and random effects to records.

The following (co)variance structure was assumed throughout:

$$V(\mathbf{u}) = \mathbf{G};$$

$$V(\mathbf{e}) = \mathbf{R};$$

$$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$$

$$V(\mathbf{Y}) = \mathbf{V} = \mathbf{ZGZ}' + \mathbf{R}.$$

The mixed model equations corresponding to this are then (Henderson, 1973):

$$\begin{bmatrix} \mathbf{X}'\mathbf{X} & \mathbf{X}'\mathbf{R}^{-1}\mathbf{Z} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{X} & \mathbf{Z}'\mathbf{R}^{-1}\mathbf{Z}+\mathbf{G}^{-1} \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{u}} \end{bmatrix} = \begin{bmatrix} \mathbf{X}'\mathbf{R}^{-1}\mathbf{Y} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{Y} \end{bmatrix} \quad \underline{\text{or}} \quad \mathbf{C}\hat{\mathbf{f}} = \mathbf{r} \quad (2)$$

For a bivariate analysis between traits recorded on male and female animals, with records for an animal on a single trait only (therefore, with no environmental covariance between the traits) and a single common litter of birth effect estimated across both traits, the model can be expanded.

If we let $\mathbf{Y}' = \mathbf{y}^{*\prime}_{\male} + \mathbf{y}^{*\prime}_{\female}$; where $\mathbf{y}^{*\prime}_{\male} = (\mathbf{y}_{\male}\ \mathbf{0})$ and $\mathbf{y}^{*\prime}_{\female} = (\mathbf{0}\ \mathbf{y}_{\female})$; the matrices and vectors used in the mixed model equations can be extended to give:

$$\mathbf{Y} = \begin{bmatrix} \mathbf{y}_{♂} \\ \mathbf{y}_{♀} \end{bmatrix}; \qquad \mathbf{e} = \begin{bmatrix} \mathbf{e}_{♂} & \mathbf{0} \\ \mathbf{0} & \mathbf{e}_{♀} \end{bmatrix}; \qquad \mathbf{Z} = \begin{bmatrix} \mathbf{Z}_{♂} & \mathbf{0} & \mathbf{Z}_{c♂} \\ \mathbf{0} & \mathbf{Z}_{♀} & \mathbf{Z}_{c♀} \end{bmatrix};$$

$$\mathbf{b} = \begin{bmatrix} \mathbf{b}_{♂} \\ \mathbf{b}_{♀} \end{bmatrix}; \qquad \mathbf{X} = \begin{bmatrix} \mathbf{X}_{♂} & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_{♀} \end{bmatrix}; \qquad \mathbf{u}' = \begin{bmatrix} \mathbf{a}_{♂}' & \mathbf{a}_{♀}' & \mathbf{c}' \end{bmatrix}$$

Where:

$\mathbf{X}, \mathbf{Z}, \mathbf{Z}_c$ - are incidence matrices relating fixed effects, random additive genetic and random litter of birth effects to records;

$\mathbf{a}_s, \mathbf{c}, \mathbf{e}_s, \mathbf{b}_s$ - are vectors of random additive genetic, litter of birth and residual effects and fixed effects for sex s, respectively;

♂,♀ - are subscripts to identify whether the matrix is associated with male or female test records.

$$V(\mathbf{u}) = \mathbf{G} = \begin{bmatrix} \mathbf{A}\sigma^2_{a♂} & \mathbf{A}\sigma_{a♂♀} & \mathbf{0} \\ \mathbf{A}\sigma_{a♂♀} & \mathbf{A}\sigma^2_{a♀} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma^2_c \end{bmatrix};$$

$$\mathbf{G}^{-1} = \begin{bmatrix} \mathbf{A}^{-1}\alpha_{♂} & \mathbf{A}^{-1}\alpha_{♂♀} & \mathbf{0} \\ \mathbf{A}^{-1}\alpha_{♂♀} & \mathbf{A}^{-1}\alpha_{♀} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}\lambda_c \end{bmatrix};$$

$\mathbf{A}$ - is the numerator relationship matrix;

$$V(\mathbf{e}) = \mathbf{R} = \begin{bmatrix} \mathbf{I}\sigma^2_{e♂} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma^2_{e♀} \end{bmatrix}; \mathbf{R}^{-1} = \begin{bmatrix} \mathbf{I}\gamma_{♂} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\gamma_{♀} \end{bmatrix};$$

$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$

$V(\mathbf{Y}) = \mathbf{V} = \mathbf{ZGZ}' + \mathbf{R}.$

The components of the MME (2), $\mathbf{C}$, $\hat{\mathbf{f}}$ and $\mathbf{r}$, can then be represented as:

$$\begin{bmatrix} \gamma_{♂}\mathbf{X}'_{♂}\mathbf{X}_{♂} & \mathbf{0} & \gamma_{♂}\mathbf{X}'_{♂}\mathbf{Z}_{♂} & \mathbf{0} & \gamma_{♂}\mathbf{X}'_{♂}\mathbf{Z}_{c♂} \\ \mathbf{0} & \gamma_{♀}\mathbf{X}'_{♀}\mathbf{X}_{♀} & \mathbf{0} & \gamma_{♀}\mathbf{X}'_{♀}\mathbf{Z}_{♀} & \gamma_{♀}\mathbf{X}'_{♀}\mathbf{Z}_{c♂} \\ \gamma_{♂}\mathbf{Z}'_{♂}\mathbf{X}_{♂} & \mathbf{0} & \gamma_{♂}\mathbf{Z}'_{♂}\mathbf{Z}_{♂}+\mathbf{A}^{-1}\alpha_{♂} & \mathbf{A}^{-1}\alpha_{♂♀} & \gamma_{♂}\mathbf{Z}'_{♂}\mathbf{Z}_{c♂} \\ \mathbf{0} & \gamma_{♀}\mathbf{Z}'_{♀}\mathbf{X}_{♀} & \mathbf{A}^{-1}\alpha_{♂♀} & \gamma_{♀}\mathbf{Z}'_{♀}\mathbf{Z}_{♀}+\mathbf{A}^{-1}\alpha_{♀} & \gamma_{♀}\mathbf{Z}'_{♀}\mathbf{Z}_{c♀} \\ \gamma_{♂}\mathbf{Z}'_{c♂}\mathbf{X}_{♂}\gamma_{♀}\mathbf{Z}'_{c♀}\mathbf{X}_{♀} & \gamma_{♂}\mathbf{Z}'_{c♂}\mathbf{Z}_{♂} & \gamma_{♀}\mathbf{Z}'_{c♀}\mathbf{Z}_{♀} & \gamma_{♂}\mathbf{Z}'_{c♂}\mathbf{Z}_{c♂}+\gamma_{♀}\mathbf{Z}'_{c♀}\mathbf{Z}_{c♀}+\mathbf{I}\lambda_{c} \end{bmatrix}$$

$$\begin{bmatrix} \hat{\mathbf{b}}_{♂} \\ \hat{\mathbf{b}}_{♀} \\ \hat{\mathbf{a}}_{♂} \\ \hat{\mathbf{a}}_{♀} \\ \hat{\mathbf{c}} \end{bmatrix} \quad \text{and} \quad \begin{bmatrix} \gamma_{♂}\mathbf{X}'_{♂}\mathbf{y}_{♂} & \mathbf{0} \\ \mathbf{0} & \gamma_{♀}\mathbf{X}'_{♀}\mathbf{y}_{♀} \\ \gamma_{♂}\mathbf{Z}'_{♂}\mathbf{y}_{♂} & \mathbf{0} \\ \mathbf{0} & \gamma_{♀}\mathbf{Z}'_{♀}\mathbf{y}_{♀} \\ \gamma_{♂}\mathbf{Z}'_{c♂}\mathbf{y}_{♂} & \gamma_{♀}\mathbf{Z}'_{c♀}\mathbf{y}_{♀} \end{bmatrix}, \text{ respectively.}$$

If the residual variance in both traits is the same ($\sigma^2_{e♂} = \sigma^2_{e♀}$) then the original Meyer algorithm can be used by setting up $\mathbf{Y}$ such that $\mathbf{Y}'=(\mathbf{y}'_{♂}\ \mathbf{y}'_{♀})$ and associating the first animal effect with records on males, and the second animal effect with records on females. However, if $\sigma^2_{e♂} \neq \sigma^2_{e♀}$ then it is necessary to rescale the model such that $\gamma_{♂} = \gamma_{♀} = 1$, and the genetic (co)variance matrix and the coefficient matrix of the mixed model equations are scaled by the appropriate residual variance. The scaled genetic (co)variance matrix, $\mathbf{G}^{*}_{a}$, is $\Sigma^{-1/2}\mathbf{G}_{a}\Sigma^{-1/2}$, where:

$$\Sigma = \begin{bmatrix} \sigma^2_{e♂} & 0 \\ 0 & \sigma^2_{e♀} \end{bmatrix} \quad \therefore \quad \Sigma^{-1/2} = \begin{bmatrix} 1/\sigma_{e♂} & 0 \\ 0 & 1/\sigma_{e♀} \end{bmatrix}$$

$$\mathbf{G}_a = \begin{bmatrix} \sigma^2_{a♂} & \sigma_{a♂♀} \\ \sigma_{a♂♀} & \sigma^2_{a♀} \end{bmatrix}$$

Which gives:

$$\mathbf{G}^*_a = \begin{bmatrix} \sigma^2_{a♂}/\sigma^2_{e♂} & \sigma_{a♂♀}/\sigma_{e♂}\sigma_{e♀} \\ \sigma_{a♂♀}/\sigma_{e♂}\sigma_{e♀} & \sigma^2_{a♀}/\sigma^2_{e♀} \end{bmatrix}$$

$$\mathbf{G}^{*-1}_a = \phi \begin{bmatrix} h^2_{♀}/e^2_{♀} & -h_{♂♀}/e_{♂}e_{♀} \\ -h_{♂♀}/e_{♂}e_{♀} & h^2_{♂}/e^2_{♂} \end{bmatrix} = \begin{bmatrix} \alpha_{♂} & \alpha_{♂♀} \\ \alpha_{♂♀} & \alpha_{♀} \end{bmatrix}$$

Where: $h^2_{♂}, h^2_{♀}$ are the heritabilities of the traits in males and females;

$e^2_{♂}, e^2_{♀}$ are the residual variances as a proportion of the phenotypic variances;

$h_{♂♀}$ is the coheritability, $\sigma_{a♂♀}/\sigma_{p♂}\sigma_{p♀}$, (Yamada, 1968) of the two traits;

ϕ $= e^2_{♂}e^2_{♀} / (h^2_{♂}h^2_{♀} - h^2_{♂♀})$.

The matrix **C** is scaled such that the scaled coefficient matrix of the mixed model equations, $\mathbf{C}_S$, is equal to $\mathbf{S}_C\mathbf{C}\mathbf{S}_C$; where:

$$\mathbf{S}_C = \begin{bmatrix} \mathbf{I}_1\sigma_{e♂} & 0 & 0 & 0 & 0 \\ 0 & \mathbf{I}_2\sigma_{e♀} & 0 & 0 & 0 \\ 0 & 0 & \mathbf{I}_3\sigma_{e♂} & 0 & 0 \\ 0 & 0 & 0 & \mathbf{I}_3\sigma_{e♀} & 0 \\ 0 & 0 & 0 & 0 & \mathbf{I}_4\sigma_{e♂} \end{bmatrix};$$

and $\mathbf{I}_1$, $\mathbf{I}_2$, $\mathbf{I}_3$ and $\mathbf{I}_4$ are identity matrices with ranks equal to the number of

levels of fixed effects on the male trait, number of levels of fixed effects on the female trait, number of animals and number of litters of birth, respectively. The scaling for the common litter of birth effect, which is assumed to have a unit between sex correlation, has been arbitrarily scaled by the residual variance of the male trait and is expressed relative to this trait, that is $c^2 = \sigma^2_c/\sigma^2_{p\sigma}$. After this **R** is an identity matrix and **G** is:

$$\mathbf{G} = \begin{bmatrix} \mathbf{A}\otimes\mathbf{G}^*_a & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma^2_c/\sigma^2_{e\sigma} \end{bmatrix}$$

Where $\otimes$ is the direct (Kronecker) product as defined in Searle (1966). The numerator relatationship matrix , **A**, for each Test Management Group was based on pedigrees going back to the beginning of the data collection period (that is the earliest records in Test Management Group 1).

From Meyer (1989a) the likelihood function can be written as:

$$-2 \log L = \text{constant} + \log|\mathbf{R}| + \log|\mathbf{G}| + \log|\mathbf{C}^*_S| + \mathbf{Y'PY}$$

where $\mathbf{C}^*_S$ is a full rank submatrix of $\mathbf{C}_S$ (**X** replaced by full rank submatrix $\mathbf{X}^*$) and $\mathbf{P} = \mathbf{V}^{-1} - \mathbf{V}^{-1}\mathbf{X}^*(\mathbf{X}^{*\prime}\mathbf{V}^{-1}\mathbf{X}^*)^{-1}\mathbf{X}^{*\prime}\mathbf{V}^{-1}$. For the scaled bivariate model, $\mathbf{V} = \mathbf{S}\mathbf{V}_S\mathbf{S}$; with $\mathbf{S} = \begin{bmatrix} \mathbf{I}\sigma_{e\sigma} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma_{e\varphi} \end{bmatrix}$. Substituting this into **Y'PY** gives:

$$\mathbf{Y'PY} = \mathbf{s}' \begin{bmatrix} \mathbf{y}^{*\prime}_{\sigma}\mathbf{P}_S\mathbf{y}^*_{\sigma} & \mathbf{y}^{*\prime}_{\sigma}\mathbf{P}_S\mathbf{y}^*_{\varphi} \\ \mathbf{y}^{*\prime}_{\varphi}\mathbf{P}_S\mathbf{y}^*_{\sigma} & \mathbf{y}^{*\prime}_{\varphi}\mathbf{P}_S\mathbf{y}^*_{\varphi} \end{bmatrix} \mathbf{s}$$

where $\mathbf{s}' = [1/\sigma_{e\sigma}\ 1/\sigma_{e\varphi}]$ and $\mathbf{P}_S = \mathbf{V}^{-1}_S - \mathbf{V}^{-1}_S\mathbf{X}^*(\mathbf{X}^{*\prime}\mathbf{V}^{-1}_S\mathbf{X}^*)^{-1}\mathbf{X}^{*\prime}\mathbf{V}^{-1}_S$.

The log determinant of $\mathbf{C}^*_S$ was accumulated during the Gaussian

elimination step and the other components of the likelihood were calculated as:

$$\log |\mathbf{R}| = (n_{rec\,♂} - n_{f\,♂}) \log \sigma^2_{e♂} + (n_{rec\,♀} - n_{f\,♀}) \log \sigma^2_{e♀}$$

$$\log |\mathbf{G}| = n_{anim} \left\{ \log \frac{\sigma^2_{a♂}}{\sigma^2_{e♂}} + \log \left(\sigma^2_{a♀} - \frac{\sigma_{a♂a♀}\,\sigma_{a♂a♀}}{\sigma^2_{a♂}} \right) \frac{1}{\sigma^2_{e♀}} \right\}$$

$$+ n_c \log \frac{\sigma^2_c}{\sigma^2_{e♂}}$$

$$\mathbf{Y'PY} = \frac{ypy_{♂♂}}{\sigma^2_{e♂}} + \frac{2ypy_{♂♀}}{\sigma_{e♂}\sigma_{e♀}} + \frac{ypy_{♀♀}}{\sigma^2_{e♀}}$$

Where:

n_{anim} - is the number of animals in the analysis;

n_{reci} - is the number records for trait i;

n_{fi} - is the total numbers of fixed effect levels for trait i;

n_c - is the number of levels of the common litter of birth effect;

ypy_{ij} - are residual sums of squares, or cross-products.

For a given set of values of $\alpha_{♂}$, $\alpha_{♂♀}$, $\alpha_{♀}$ and λ_c, the likelihood is a constant plus:-

$$\left[n_{df♂} \log \sigma^2_{e♂} + n_{df♀} \log \sigma^2_{e♀} + \frac{ypy_{♂♂}}{\sigma^2_{e♂}} + \frac{2ypy_{♂♀}}{\sigma_{e♂}\sigma_{e♀}} + \frac{ypy_{♀♀}}{\sigma^2_{e♀}} \right];$$

where n_{df_i} is the number of degrees of freedom for trait i. Differentiation of this function with respect to $\sigma^2_{e♂}$ and $\sigma^2_{e♀}$ and equating to zero gives:-

$$\frac{df_{♂}}{\sigma_{e♂}} = \frac{ypy_{♂♂}}{\sigma^3_{e♂}} + \frac{ypy_{♂♀}}{\sigma^2_{e♂}\sigma_{e♀}} \quad \Rightarrow \quad df_{♂}\sigma^2_{e♂} = ypy_{♂♂} + x\, ypy_{♂♀};$$

$$\frac{df_{♀}}{\sigma_{e♀}} = \frac{ypy_{♂♀}}{\sigma_{e♂}\sigma^2_{e♀}} + \frac{ypy_{♀♀}}{\sigma^3_{e♀}} \quad \Rightarrow \quad df_{♀}\sigma^2_{e♀} = \frac{1}{x} ypy_{♂♀} + ypy_{♀♀};$$

where $x = \sigma_{e♂}/\sigma_{e♀}$. Values of $\sigma^2_{e♂}$ and $\sigma^2_{e♀}$, are calculated by iterating on the two equations given above.

The analysis described above is equivalent to fitting a maternal genetic effect, with a single residual variance, in the univariate case (see Chapter 2; model 3 or Meyer,1989a). This method is equivalent to an individual animal model specific case of the methodology given by Schaeffer, Wilton and Thompson (1978). The mixed model equations for these analyses are therefore similar to theirs, making use of a relationship matrix and augmenting the equations by the terms required for estimation of a single common litter of birth effect across the sexes.The maximum of the log likelihood (L) function was evaluated using a Simplex procedure, the variance of the values of -2L stored in the Simplex being used to define convergence. The convergence criterion used was $var(-2L) = 1x10^{-7}$.

RESULTS

Results of between sex bivariate analyses within each breed and test management group are given in tables 3.2 and 3.3. Phenotypic means of the traits can be found in the table 2.3 (Chapter 2).

Estimates of the genetic correlations between the sexes are consistently high across traits, test management groups and breeds. There are two exceptions to this; the estimates for ADFI and FCR in the second and fourth Large White Test Management Groups which had values of 0.26 and 0.51, respectively. In both of these cases the additive genetic variance in females is very low, limiting the covariance to being close to zero. Approximate standard errors of the genetic correlations between male and female ADFI in LW TMG 2 and between male and female FCR in LW TMG 4

were found to be 0.24 and 0.19, respectively, using the formula given by Falconer (1981).

DISCUSSION

The analysis reported here could be regarded in one of two ways; either as an investigation of genotype-environment interaction between two groups of pigs tested under different regimes, or as an investigation of similarities in the genetic control of performance test traits between male and female pigs. In these two cases differences due to either the sex or the environment are ignored. The results are of course due to a combination of both of these situations.

The current results could be said to be in agreement with the results of Merks (1986) and Ollivier (1983) with regard to genotype-sex interactions. With hindsight, it is easy to categorise the test regimes in males and females as being very similar and to describe the results as being predictable given previous results, in particular those of Ollivier. However, the data on ADFI and days on test presented in Chapter 2 showed that there were noticeable differences between the test undergone by boars and gilts, and the within sex univariate analyses produced noticeably different estimates of the variance components.

If the effect of genotype-sex interactions upon the genetic correlation estimates are ignored, and the males and females are regarded as being under different management regimes then they are in accordance with the results of Van Diepen and Kennedy (1990), but not with those of Merks for GxE across breeding pyramid levels.

Table 3.2: Results of between sex bivariate analyses of Landrace performance test data.

Trait	TMG*	$\sigma^2_{e♂}$	$\sigma^2_{e♀}$	$\sigma^2_{p♂}$	$\sigma^2_{p♀}$	$h^2_{♂}$	$h^2_{♀}$	$r_{g♂♀}$	c^2
ShFat	1	5.44	6.06	7.63	9.19	0.25	0.31	0.89	0.03
	2	5.52	6.51	7.70	8.32	0.25	0.23	0.79	0.03
	3	7.28	6.25	9.73	8.00	0.22	0.19	1.00	0.03
LFat	1	1.57	1.76	2.08	2.54	0.20	0.25	0.85	0.05
	2	1.64	1.68	1.98	1.97	0.12	0.10	0.73	0.05
	3	2.08	1.66	2.24	2.05	0.05	0.16	1.00	0.03
CFat	1	1.33	1.38	2.06	2.46	0.30	0.39	0.81	0.06
	2	1.35	1.29	2.12	1.85	0.30	0.24	0.98	0.07
	3	1.31	0.99	2.07	1.85	0.35	0.44	1.00	0.02
KFat	1	1.37	0.38	2.17	2.53	0.32	0.40	0.81	0.05
	2	1.34	1.32	2.27	1.95	0.36	0.27	0.94	0.06
	3	1.37	1.05	2.13	1.89	0.33	0.41	1.00	0.03
ADFI	1	0.0148	0.0048	0.0206	0.0055	0.23	0.09	0.89	0.05
	2	0.0189	0.0119	0.0257	0.0168	0.16	0.19	0.98	0.10
	3	0.0225	0.0106	0.0296	0.0126	0.15	0.07	1.00	0.09
WtG	1	15.1	8.1	19.6	9.6	0.19	0.12	0.84	0.04
	2	16.1	9.9	19.0	13.8	0.07	0.20	0.97	0.08
	3	21.8	13.1	26.1	16.9	0.11	0.17	0.95	0.06
ADG	1	0.00589	0.00227	0.00771	0.00270	0.20	0.12	0.82	0.04
	2	0.00617	0.00396	0.00728	0.00552	0.07	0.20	0.95	0.08
	3	0.00842	0.00411	0.01052	0.00534	0.10	0.17	0.94	0.06
FCR	1	0.0175	0.0228	0.0256	0.0294	0.27	0.18	1.00	0.04
	2	0.0213	0.0250	0.0284	0.0346	0.21	0.24	1.00	0.04
	3	0.0246	0.0288	0.0296	0.0397	0.11	0.22	1.00	0.06

* Test Management Group as described in table 3.1

Table 3.3: Results of between sex bivariate analyses of Large White performance test data.

Trait	TMG*	$\sigma^2_{e♂}$	$\sigma^2_{e♀}$	$\sigma^2_{p♂}$	$\sigma^2_{p♀}$	$h^2_{♂}$	$h^2_{♀}$	$r_{g♂♀}$	c^2
ShFat	1	7.85	7.08	11.4	9.0	0.27	0.18	0.86	0.04
	2	8.30	6.95	10.6	10.7	0.19	0.33	1.00	0.03
	3	5.75	8.51	12.0	11.9	0.49	0.25	0.75	0.03
	4	8.30	11.7	9.9	13.0	0.16	0.09	0.99	0.01
LFat	1	2.22	2.12	2.91	3.12	0.18	0.26	0.97	0.06
	2	1.86	1.92	2.53	2.59	0.25	0.25	0.84	0.01
	3	1.84	1.92	2.40	2.54	0.23	0.24	0.84	0.00
	4	1.41	2.40	2.45	2.79	0.39	0.10	0.95	0.04
CFat	1	1.54	1.72	2.56	2.79	0.37	0.36	0.90	0.03
	2	1.64	1.47	2.96	3.10	0.42	0.50	0.80	0.02
	3	1.02	1.36	2.47	2.66	0.56	0.47	0.97	0.02
	4	1.59	2.11	2.22	2.43	0.28	0.12	0.89	0.01
KFat	1	1.53	1.84	2.69	3.00	0.42	0.37	0.89	0.02
	2	1.60	1.44	2.95	3.19	0.45	0.53	0.86	0.01
	3	1.22	1.36	2.59	2.66	0.50	0.46	0.98	0.03
	4	1.66	2.21	2.35	2.54	0.28	0.11	0.81	0.02
ADFI	1	0.0176	0.00613	0.0226	0.00848	0.15	0.20	1.00	0.08
	2	0.0133	0.00459	0.0236	0.00528	0.39	0.08	0.26	0.05
	3	0.0179	0.0155	0.0250	0.0186	0.28	0.17	0.91	0.00
	4	0.0137	0.0157	0.0319	0.0166	0.57	0.05	0.78	0.00
WtG	1	16.5	9.5	22.3	11.9	0.21	0.16	0.83	0.05
	2	11.2	6.5	20.3	8.0	0.41	0.14	0.71	0.04
	3	14.4	10.8	23.0	15.9	0.33	0.28	0.60	0.04
	4	22.2	21.9	24.7	23.9	0.10	0.08	0.90	0.01
ADG	1	0.00663	0.00259	0.00892	0.00326	0.21	0.16	0.85	0.05
	2	0.00437	0.00193	0.00792	0.00238	0.41	0.15	0.71	0.04
	3	0.00563	0.00444	0.00887	0.00646	0.33	0.28	0.62	0.04
	4	0.00850	0.00695	0.00948	0.00769	0.08	0.08	0.93	0.02
FCR	1	0.0205	0.0173	0.0282	0.0266	0.19	0.27	0.78	0.08
	2	0.0092	0.0151	0.0141	0.0222	0.31	0.28	0.90	0.04
	3	0.0208	0.0196	0.0259	0.0337	0.12	0.34	1.00	0.08
	4	0.0170	0.0431	0.0263	0.0469	0.35	0.08	0.51	0.00

* Test Management Group as described in Table 3.1

Apart from the two exceptions noted in the results section, the genetic correlations between sexes for the traits analysed are consistently high, across all time periods and both breeds. These results show that, in the population studied, selection on males and females separately, using different selection indices and test regimes, would not be expected to have a very great effect on rates of genetic progress. While this is very reassuring, it must be remembered that the estimates are population specific and may not apply to situations in which greater differences between male and female test regimes exist, such as when females are group fed and individual measurements of ADFI and weight gains are not recorded.

The low genetic correlation estimates for ADFI in LW TMG 2 and FCR in LW TMG 4 are probably due to genotype-environment interaction, rather than genotype-sex interaction. The male and female environments in LW Test Management Group 2 were very different, the food intake of the females being far more restricted than that of males (see figure 2.4, chapter 2). In the final Large White TMG there may have been a loss of appetite in infected gilts leading to the same effect as severe restriction. This effect could have been compounded by the herd management, since the feeding scales were reduced, by an unknown amount at an unknown time, during the last period to avoid the food wastage which occurred due to loss of appetite (thereby restricting the healthier animals). It would be expected that GxE would be more likely to be observed for ADFI and FCR in a situation where differences in feeding level define the different environments.

Cameron *et al.* (1988) found no significant evidence for a sire-feeding regime interaction in LW or LR breeds although a between feeding regime

genetic correlation of 0.42 (standard error = 0.5) for FCR was produced for the Landrace breed. Cameron (1991) found between feeding regime genetic correlation estimates of between 0.29 and 0.92 across traits, concluding that there was genotype-environment interaction for growth rate only, although results for ADFI and FCR were not presented. The results from the present study are in broad agreement with those of Cameron *et al.* (1988) and Cameron (1991); remembering that the environments in this study do not vary as much with regard to food intake as those of Cameron *et al.* or Cameron (had *ad libitum* and resticted groups), and are confounded with sex.

SUMMARY AND CONCLUSIONS.

The genetic correlations between performance test traits recorded in boars and gilts under different test regimes were estimated using a bivariate DFREML procedure.

The results indicate that the levels of genotype-environment and genotype-sex interaction between the boars and gilts on performance test were low across most traits and Test Management Groups, with all but two genetic correlation estimates lying between 0.60 and 1.0. However, the two low estimates of the genetic correlations which were observed when the environments appeared to differ most, indicate that GxE interactions may be a problem in populations where males and females are subject to test regimes with greater differences than those seen here.

The agreement of these results with those of Merks (1986) and Ollivier (1983) may indicate that the methodology which they used to estimate the genetic correlations between traits recorded in different environments was

adequate, and hence that the disagreement between the results of Van Diepen and Kennedy (1990) and the previous authors studies is due to differences between the North American and European populations studied, as proposed by Van Diepen and Kennedy.

CHAPTER 4.

BIVARIATE ESTIMATION OF GENETIC PARAMETERS FOR PERFORMANCE TEST TRAITS OF LANDRACE PIGS TESTED IN A COMMERCIAL NUCLEUS HERD.

INTRODUCTION

With the increased interest in Best Linear Unbiased Prediction in the pig breeding industry coupled with the desire to select upon more than one trait simultaneously, the need has grown for accurate estimates of correlations between traits for specific populations, both for input into BLUP algorithms and for combining breeding value estimates in an index. As pointed out by Groeneveld (1991), it is logical to approach estimation of parameters and breeding values with the same statistical model, and as the preferred model for BLUP procedures is the multivariate individual animal model, because it takes account of all relationships and combines information from all available sources, this should be used in (co)variance estimation also.

However, multivariate (co)variance parameter estimation is very expensive in terms of the computer time and memory required. The size of the problem increases with the number of random effects to be estimated, there being t(t+1)/2 (co)variances to estimate for each random effect of a t trait multivariate analysis. For multivariate analyses of traits with equal design matrices (that is, the same fixed and random effect models applied to all traits) it has been proposed that a canonical transformation of the data could be used to reduce the number of parameters to be estimated, by reduction of the analysis to a series of univariate problems (Thompson,

1976; Meyer, 1985). These procedures assume that there are only two random effects to be estimated and, from the results in chapter 2, it can be seen that for most of the Landrace data sets to be analysed here there should be three random effects included in the model (additive genetic, common litter of birth and residual).

In this case Thompson and Hill (1990) have proposed an algorithm which uses transformation of the sum of two uncorrelated random effect (co)variance matrices (e.g. common litter of birth and residual) to an identity matrix, and the additive genetic (co)variance matrix to a diagonal matrix, and have shown its high efficiency in one particular case. This algorithm allows the use of a univariate package, such as that of Meyer (1989a), to estimate correlations between traits with equal design matrices.

An alternative, perhaps simpler, scheme is to estimate the covariances between traits x and y in a bivariate analysis of x and y. For t traits this requires p(p-1)/2 sets of bivariate analyses. The population under study can be split into data sets defined by test regime and health status of the herd. Thus, changes in correlation estimates across these environments can be observed.

One of the changes involved in the population studied here was an increase in the feed allowance, so that the average daily food intake on test increased. Differences between correlations for average daily gain with food intake and food conversion ratio (FCR) under different feed regimes have been reported by Wyllie *et al.* (1979), who also give a summary of correlation estimates from previous studies under *ad libitum* and restricted feeding. Genetic correlations between gain and FCR were negative in both *ad libitum* and restricted fed populations, but the magnitude of the

correlations was higher in those studies where the animals were under restriction. For studies where both food conversion efficiency (FCE) and FCR were analysed, the correlations of gain with FCE were positive and of the same approximate magnitude as the correlations between gain and FCR. Correlations between gain and food intake were positive in both regimes, and higher in the studies on *ad libitum* fed populations. Cameron *et al.* (1988) also reported differences between genetic and phenotypic correlation estimates from *ad libitum* and restricted fed Large White pigs for average daily gain and food conversion ratio, while Gu *et al.* (1989a) concluded that differences in genetic correlation estimates between average daily gain and ultrasonic backfat depths from male and female pigs could be due to differences in the levels of feeding between the sexes.

The second major change during the data collection period was a decline in the health status of the herd. No estimates of genetic correlations between traits in herds of different health status could be found, but Kennedy and Quinton (1987) showed that there were genetic and environmental differences between herds with different health classifications, although the genetic differences could have been a result of different selection pressures being applied to traits in different environments.

MATERIAL AND METHODS

Data and population structure:

The data analysed were performance test data collected over a six year period in a single purebreeding Landrace nucleus population. The details of the performance test differed between the sexes and changed

during the data collection period, but the test can broadly be described as being over a weight range of 40-90kg (approximately 3-5 months of age), with an increasing feeding scale during the test. All animals finishing test in a given week were assigned to the same test batch, and during the analysis a test month was regarded as being a group of four consecutive test batches. As a result of changes in the test regime, the data were divided into three Test Management Groups within each sex. Details of the test and Test Management Groups are given in Chapter 2.

The traits to be analysed were total weight gain on test {WtG; recorded in kg}, average daily gain during the test {ADG; kg/day}, average daily food intake (ADFI=total amount of food consumed during test/days on test; kg/day), food conversion ratio (FCR=ADFI/ADG; kg intake/kg gain) and four ultrasonic fat depths (measured at the shoulder {Sh Fat}, loin {L Fat} and approximate P2 and P3 positions {C Fat and K Fat}; all recorded in mm). All traits were recorded on both sexes.

Analyses performed:

Each data set was analysed using the univariate Derivative-Free REML (DFREML) algorithm developed by Dr. K. Meyer. This algorithm is an extension of the method proposed by Smith and Graser (1986, and Graser *et al.*,1987) to allow simultaneous estimation of multiple random effects (Meyer,1989a).

Thompson and Hill (1990) showed how correlations between traits with identical design matrices could be estimated using a univariate algorithm by analysis of individual traits and then sums of pairs of traits, or a transformation, $\mathbf{z}=\mathbf{H}_0\mathbf{y}$, where $\mathbf{y}$ is a matrix of observations , $\mathbf{z}$ is a matrix of

observations on the transformed scale and $\mathbf{H}_0$ is a transformation matrix whose elements are zeroes and ones defining traits and combinations of pairs of traits. The covariance between two traits, x and y, is estimated as:

$$\text{cov}(x,y) = 0.5\{ \text{var}(x+y) - \text{var}(x) - \text{var}(y)\}$$

Use of this formula across different random effects (additive genetic, common litter of birth and residual in this case) gives estimates for the covariances between traits for each effect included in the analysis. From these a transformation, **T**, can be calculated to transform the matrix $\mathbf{V}_T$ (where $\mathbf{V}_T = \mathbf{V}_E + \mathbf{V}_C$, and $\mathbf{V}_E$ and $\mathbf{V}_C$ are the residual and common environmental (co)variance matrices) to an identity matrix and the additive genetic (co)variance matrix, $\mathbf{V}_A$, to a diagonal matrix, $\mathbf{V}_A^*$, the elements of which are the eigenvalues of $\mathbf{V}_A$. New variables, which are less correlated, can then be generated from $\mathbf{z} = \mathbf{H}_0\mathbf{T}\mathbf{y}$, and the univariate analysis procedure repeated. Back transformation of variances to the original 'y' variable scale allows estimation of a revised **T**, and the cycle can be repeated until the transformed 'z' variables are approximately uncorrelated. In each successive round of univariate analyses the transformation is expected to take better account of the covariances between the traits, and the subsequent new covariance estimates are expected to improve.

For the example presented by Thompson and Hill, the transformed traits were essentially uncorrelated after 3 cycles of covariance estimation. It was observed during preliminary runs with these data that the genetic and phenotypic correlations between transformed traits were also very low after 3 cycles and that there was little change in (co)variance estimates if the procedure was allowed to go beyond this point. Therefore, the results presented here were produced by three cycles of univariate analyses.

In order to perform this analysis across all six data sets and all combinations of test traits as efficiently as possible, the above algorithm was incorporated into the existing analysis program, transforming the submatrix of the mixed model array which contained the residual sums of squares and cross-products of traits after Gaussian elimination, rather than the data itself, each time the likelihood function was evaluated. Subroutines were included to transform variance-covariance matrices to give estimates on the original scale.

Although it is theoretically possible to perform a single multivariate analysis of all traits using this algorithm, because traits are initially considered as pairs (therefore no account is taken of covariances with other traits) and some of the traits are highly correlated, it was observed in preliminary runs that the analysis failed due to the production of negative definite (co)variance matrices. Therefore, a series of bivariate analyses were performed within each Test Management Group.

Each univariate analysis was performed fitting the following Individual Animal model (model 1):

$$Y_{ijklmn} = a_i + MT_j + b1.L_{ijkn} + b2.W_{ijln} + b3.D_{ijmn} + c_n + e_{ijklmn}$$

Where:

Y_{ijklmn} - is the phenotypic record of individual i for the trait;

a_i - is the random additive genetic effect of the i^{th} individual;

MT_j - is the fixed effect associated with the j^{th} month of test;

$b1.X_{ijkn}$ - is the partial regression of Y on length of test period (days);

$b2.X_{ijln}$ - is the partial regression of Y on weight at start of test (kg);

$b3.X_{ijmn}$ - is the partial regression of Y on age at start of test (days);

c_n - is the random effect of the n^{th} litter of birth;

e_{ijklmn} - is the random environmental effect associated with the record, Y_{ijklmn}.

The expectations of the additive direct genetic effect (a_i), common litter of birth effect (c_n) and residual error effect (e_{ijklmn}) are zero, and their variances are σ_a^2, σ_c^2 and σ_e^2 respectively.

From univariate analyses assessing different random effects, it was clear that there was either no common litter of birth effect or an inadequate data structure to allow its estimation in Landrace boar Test Management Groups 2 and 3. Subsequently, the data from these groups were analysed both under model 1, including a common litter of birth effect, and under a second model from which this effect was omitted (model 2); thus:

$$Y_{ijklm} = a_i + MT_j + b1.X_{ijk} + b2.X_{ijl} + b3.X_{ijm} + e_{ijklm}$$

where all symbols are as defined for model 1.

The linear model can also be represented in matrix notation as:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e}$$

Where:

Y - is a vector of n_{rec} observations;

b - is a vector of n_f (where: $n_f=n_m+3$; n_m as in table 2.1) fixed month of test effects and linear covariates;

X - is an n_{rec} x n_f incidence matrix relating fixed effects to records;

Z - is an n_{rec} x n_u incidence matrix relating random effects to records;

u - is a vector of n_u random effects;

$\mathbf{u}' = (\mathbf{a}' \ \mathbf{c}')$ for model 1, and

$\mathbf{u}' = \mathbf{a}'$ for model 2;

a,c - are vectors of n_{anim} and n_c random additive genetic and common litter of birth effects, respectively;

The following variance structure was assumed throughout:

$V(\mathbf{u}) = \mathbf{G}$;

that is, $\mathbf{G} = \begin{bmatrix} \sigma_a^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$ for model 1, and $\mathbf{G} = \sigma_a^2\mathbf{A}$ for model 2;

$V(\mathbf{e}) = \mathbf{R} = \sigma_e^2\mathbf{I}$;

$cov(\mathbf{u},\mathbf{e}') = 0$;

$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}$.

RESULTS

Means of the traits within each Test Management Group are given in table 2.4 (Chapter 2). Univariate estimates of parameters and residual and phenotypic variances appear in tables 2.9.i - 2.9.vi, Variance components will not be presented for each analysis performed here.

Estimates of genetic and phenotypic correlations between traits for boars and gilts analysed with model 1 appear in tables 4.1 and 4.2, respectively, while residual and common litter of birth correlations from these analyses are in tables 4.3 and 4.4. Because of the similarity between bivariate and univariate variance parameter estimates, and their consistency across analyses, only estimates from the first boar Test Management Group are presented (table 4.5). Results from analyses of Landrace boar Test Management Groups 2 and 3 under model 2 appear in table 4.6 (correlations and univariate estimates of the heritability only).

Within these tables "-var" indicates a missing result caused by a negative variance component estimate for one of the traits (on the original scale). The "Fail" entry in the tables corresponds to an analysis for which no canonical transformation could be estimated because the matrix $\mathbf{V}_T$ was negative definite (for a bivariate analysis this is equivalent to an absolute value of the correlation greater than one).

Analyses containing common litter of birth effect (model 1)

Genetic correlations, r_g

Estimates of r_g between the four ultrasonic fat measurements are generally positive and reasonably high (only two estimates below 0.35 from boar data sets, and one below 0.4 from gilt data sets). The genetic correlation between C Fat and K Fat across all Test Management Groups is approximately one.

Estimates of r_g between fat measurements and WtG and ADG are generally negative, with the gilt estimates being of greater magnitude. In the third boar Test Management Group, positive correlations are observed.

Genetic correlations between ADFI and the ultrasonic fat depths are positive across all Test Management Groups and are highest in the second TMG. In the third TMG the estimate of r_g between Sh Fat and ADFI was greater than one. The corresponding estimates from gilt TMGs are less consistent than those from boars, the correlation of ADFI with Sh Fat is lower in TMG 1 than in the later Test Management Groups, whilst that with C Fat declined across TMGs. Negative values of r_g were found for ADFI with L Fat (in gilt TMGs 2 and 3), and with K Fat (gilt TMG 3).

Total weight gain and average daily gain on test are always positively

genetically correlated with ADFI in boar data sets, with the lowest estimates from TMG2. However, estimates of these r_gs from gilt TMG1 are negative, and the magnitude of the estimates in TMG3 are considerably less than the contemporary boar estimates.

In both males and females the genetic correlation between ADFI and FCR increased from TMG1 to TMG2, and subsequently decreased in TMG3 (the boar estimate in TMG3 is slightly negative).

Common litter of birth correlations, r_c

Estimates of the common litter of birth correlations between the performance test traits in boar Test Management Groups 2 and 3 are poor, many of the absolute values exceeding one. The trends across groups shall only be described in terms of the estimates sign and magnitude (relative to r_c estimates from other Test Managment Groups), no standard error estimates were calculated.

Changes in the common litter of birth correlations were observed across gilt Test Management Groups for WtG, ADG, FCR and ADFI with the backfat measures. In particular, WtG and ADG were negatively correlated with backfat depths in the first gilt TMG only, while ADFI had low correlations (of both signs) with backfat depths in the first TMG and the correlations between FCR and the backfat measures changed evenly from one positive to negative across the three TMGs. There was some indication of changes in magnitude, rather than sign, of these correlations across boar Test Management Groups.

Food conversion ratio had a low negative correlation with ADFI in gilt TMGs 1 and 3, and moderate positive correlations in the second gilt TMG.

FCR was highly positively correlated with ADFI in all boar TMGs.

Phenotypic and residual correlations, r_p and r_e

Phenotypic and environmental correlations between traits are consistent across Test Management Groups in boars, while estimates from gilt TMG1 differ from those in the following periods.

Variance parameters, h^2 and c^2

Estimates of heritabilities and common litter of birth effects change very little from their univariate estimates (see chapter 2, or diagonal elements of correlation tables 4.1 - 4.4).

Analyses omitting common litter of birth effect (model 2)

Estimates of the phenotypic and environmental correlations under model 2 were very similar to those from model 1, and heritability estimates differed little from their univariate estimates.

Genetic correlation estimates under model 2 differed from those under model 1, although the signs and trends across the two Test Management Groups were generally similar. Changes in sign of correlation estimates occurred for WtG and ADG versus C Fat and K Fat in TMG 2, and for Sh Fat with all of the other backfat measurements, WtG and ADG in TMG 3.

DISCUSSION

Changes in correlations across Test Management Groups

Across Test Management Groups factors which have changed which are expected to affect the correlations between traits are the degree of restriction, health status, amount of selection previously applied and the halothane status of the herd.

According to Falconer (1981), continuous simultaneous selection to increase two traits will result in the genetic correlation between them becoming negative. This is because favourable pleiotropic genes will tend to fixation and subsequently not contribute to the genetic variances of the traits or the covariance between them, whilst genes having a favourable effect in one trait and an unfavourable one in the other will remain unfixed. This effect could not be observed across the three consecutive time periods which make up the Test Management Groups, the data collection period being too short for there to be differences between the TMGs.

The degree of restriction applied in the herd was expected to have an effect upon the genetic correlations between the growth traits (ADFI, WtG, ADG and FCR) and the measures of backfat. This effect has been observed by Gu *et al.* (1989a) and is due to the differences in partitioning of energy into lean and fat at different levels of restriction. Lean growth requires less energy than fat deposition and so at higher levels of restriction the animals which grow faster will be those which are laying down muscle rather than fat, while at less restricted feeding levels energy is not the limiting factor and so the animals which grow faster will be producing both lean and fat. This theory is the basis of much of the performance testing which has been

carried out in Europe over the past 20 years (Kielanowski, 1968; Fowler, Bichard and Pease, 1976), with animals fed on a retricted scale in order that the animals genetically inclined toward lean deposition may be identified.

As a result of this it was expected that that under restricted conditions (in particular, the first Test Management Group in gilts) ADFI and fat depths would be genetically uncorrelated, while the measures of weight gain would be negatively genetically correlated with fat depths and positively genetically correlated with ADFI. Positive genetic correlations are expected between all three combinations of traits under *ad libitum* conditions.

Considering the genetic correlation estimates produced from gilt TMGs 1 and 2 (in which the feeding scale level is the main difference), the results are not as expected. Rather than the pattern of results expected for a restricted population being displayed in TMG 1, ADFI and weight gain are negatively correlated (although the estimates are of low magnitude) while ADFI is positively genetically correlated with backfat depth. Since it is obvious from the graphs of phenotypic variance presented in chapter 2 that the gilts in TMG 1 are highly restricted, it can only be concluded that the unexpected values of the genetic correlations between ADFI and measures of fat depth and weight gain are due to the very low magnitude of the additive genetic variance for ADFI, and will be subject to large standard errors.

In TMG 2, weight gain is positively genetically correlated with ADFI and negatively with the fat depth measures, which is indicative of this Test Management Group still being under restricted feeding. Compared to TMG 1, ADFI is generally less genetically correlated with backfat depth (indeed, there is one negative estimate between ADFI and L Fat). These estimates

may still be from a restricted group of animals, but subject to lower standard errors because of the increase in the variation in ADFI (which was of course due to the change in feeding level).

The results for genetic correlations from boar TMGs 1 and 2 are very similar (under model 1). Low negative correlations between weight gain and fat depth measures indicate that the boars may be slightly restricted, indeed the lower genetic correlations between weight gain and ADFI in TMG 2 would appear to indicate that this TMG is more restricted than the first. However, when the results for TMG 2 are taken from model 2 (no common litter of birth effect included) then it appears that there is no longer any restriction upon the boars. Generally, higher positive genetic correlations of ADFI with fat depths and positive estimates of the genetic correlations of weight gains with fat depths were observed in TMG 2 (under model 2).

Without results from the Large White breed, it is not possible to demonstrate any differences between genetic correlations caused by change in the halothane status of the population. However, the fixation of genes affecting leanness and growth rate, as the halothane gene does, will remove its contribution from both the variances and covariances of the traits. It may be expected that in a population with a high incidence of halothane sensitivity, the genetic correlation between ADG and fat depths will be negative, or at least influenced towards being negative, due to the fact that halothane sensitive pigs have lower FCR and are leaner (Webb and Simpson, 1986). In a population under restricted feeding lean growth is more efficient, requiring less energy than fat deposition, and so lean halothane reactors will grow faster in this situation.

The decline in the health status of the herd in the third Test Management Group of both sexes may be expected to alter genetic correlations among traits if there is some degree of genetic control of disease resistance, the genes involved in resistance becoming more important than genes controlling performance under 'normal' health conditions and the genetic correlations being those caused by the interaction of the resistance genes with the traits of interest.

It must be noted here that there is confounding of the change in health status with a decrease in the feeding scales offered to both sexes. The magnitude and size of the change is unknown, it was performed as a management response to the observed loss of appetite in the herd in order to minimise the amount of food being wasted. However, the result would be to restrict the animals that are inclined to consume more food, which would be expected to be those which remained healthy.

There are some differences between estimates of genetic correlations in Test Management Groups 2 and 3, in particular the genetic correlations between FCR and the weight gain traits become highly negative in TMG3 (both sexes, model 1), and the genetic correlation of FCR with ADFI decreases in magnitude (again in both sexes).

Differences in correlations between analyses under alternative random effect models

The estimates of phenotypic and residual correlations between the traits in Landrace boar Test Management Groups two and three only change marginally if the common litter of birth is omitted from the analysis. However, the estimates of the genetic correlation are considerably different.

Falconer (1981) gives the phenotypic correlation between traits for model 2 (that is with additive genetic and environmental effects only) as:

$$r_{p2} = r_{g2}h_{x2}h_{y2} + r_{e2}\sqrt{(1-h_{x2}^2)}\sqrt{(1-h_{y2}^2)}$$

where r_{p2}, r_{g2}, r_{e2} are phenotypic, genetic and environmental correlations between traits x and y, respectively, and h_{x2} and h_{y2} are the square roots of the heritabilities of these traits from model 2. By following the same arguments as in Falconer (1981), the phenotypic correlation for model 1 can be represented by:

$$r_{p1} = r_{g1}h_{x1}h_{y1} + r_{c1}c_{x1}c_{y1} + r_{e1}\sqrt{(1-h_{x1}^2-c_{x1}^2)}\sqrt{(1-h_{y1}^2-c_{y1}^2)}$$

where subscript 1 refers to the model, r_c is the common litter of birth correlation between traits x and y and c_{x1} and c_{y1} are the square roots of the common litter of birth effects, c_{x1}^2 and c_{y1}^2.

These equations can be rewritten so that:

$$r_{g1} = \{ r_p - r_{c1}c_{x1}c_{y1} - r_e\sqrt{(1-h_{x1}^2-c_{x1}^2)}\sqrt{(1-h_{y1}^2-c_{y1}^2)} \}/h_{x1}h_{y1}$$

$$r_{g2} = \{ r_p - r_e\sqrt{(1-h_{x2}^2)}\sqrt{(1-h_{y2}^2)} \}/h_{x2}h_{y2}$$

where r_p and r_e are assumed to be equal across models. From these equations it can be seen that differences in the estimates of genetic correlations between the two models can be caused by differences in the magnitude of common litter of birth and additive genetic effects for each trait, or by the size of the estimate of the common litter of birth correlation. In fact separation of the cause of the differences seen was not possible, the above factors interacting with one another. The magnitude of the heritability under the altenative models would have the largest effect, the common litter

of birth terms being very small compared to these.

Approximate standard errors of the genetic correlations under models 1 and 2 (table 4.7) for boar Test Management Groups 2 and 3 were calculated using the formula given by Falconer (1981):

$$se(r_g) = \frac{1 - r_g^2}{\sqrt{2}} \sqrt{\frac{se(h_x^2).se(h_y^2)}{h_x^2.h_y^2}}$$

Heritabilities and their standard errors were taken from the univariate analyses under these models presented in chapter 2.

Although it is not possible to test for statistical differences between the estimates of the genetic correlations under the alternative models, it can be seen from the size of the standard error estimates that it would be difficult to statistically differentiate between many of the estimates.

The difficulties observed in this section, where common litter of birth effects were found to be low, leading to a poor estimate of the correlation for this random effect, were inherent in the algorithm used. In transforming the matrix $\mathbf{V}_T = \mathbf{V}_E + \mathbf{V}_C$ to an identity matrix, when the estimates of c^2 are small, the algorithm effectively transforms $\mathbf{V}_E$ and alters $\mathbf{V}_C$ as a by-product. As a result of this, the transformed traits were still correlated with regard to the common litter of birth effect.

The algorithm of Meyer (1989a) imposes a limit on the ranges of parameter estimates, but this was not altered to take account of transforming the data and so when the estimate of a parameter was very low on the transformed scale, back transformation could produce negative variance component estimates. Similarly, correlation estimates which fell outside of the range -1 to +1 were generated if the value for the variance

parameter for the combined trait lay between zero and one. This happened most frequently when the common litter of birth variances were very low, allowing there to be too large a parameter space in which to estimate the covariance, or when traits were highly correlated.

In order to utilise the variance-covariance matrices produced here in a multivariate procedure, it may be necessary to perform some operation on them to ensure that the matrices were positive semi-definite. A bending procedure (Hayes and Hill, 1980) in which a bending factor was chosen which forced the most negative eigenvalue estimate to be positive would be suitable. The factor could be selected across (co)variance matrices for different random effects and the same transformation performed on all of the (co)variance matrices involved, thereby maintaining the additive properties of the matrices (i.e. the phenotypic (co)variance matrix after bending being the sum of the constituent matrices after bending).

As well as being used to make the estimates produced here usable in multivariate breeding value estimation, bending could be incorporated into the algorithm to allow a true multivariate analysis to be performed. It may be expected that bending would only be required in the early stages of the analysis when information on the relationships between all traits is low (covariance estimated from a series of bivariate analyses).

SUMMARY AND CONCLUSIONS

Changes in the environment caused changes in the genetic correlations between traits. As a result of increasing the food allowance, estimates of the genetic correlations between weight gain and backfat depths decreased in magnitude between gilt TMGs 1 and 2, but remained

negative, indicating that the gilts remained restricted after their feeding scale was increased. Estimates of these correlations were low and negative for boar Test Management Group 1, and were positive in TMGs 2 and 3 under model 2, any slight restriction on the boars having been removed.

The effect of reduced health status upon the genetic correlation estimates from boars (model 2) was an increase in the correlations between weight gain and backfat depths, with a related change in the genetic correlations of the backfat measures with FCR. There are increases in the estimates of ADFI versus weight gain genetic correlations, and all of these changes indicate that the health status decline has a similar effect to increasing the food allowance. This trend is repeated in the gilt estimates, except for the correlations of ADFI with weight gain. This may be due to a health induced loss of appetite throughout the herd reducing the level of restriction upon the sick animals.

Although little difference had been observed between heritability estimates under models containing or omitting common litter of birth effects for most traits in boar Test Management Groups 2 and 3 (chapter 2), it was observed that the genetic correlation was sensitive to estimation of an effect which either did not exist or could not be estimated. This demonstrates the importance of selection of suitable models, which, given computing considerations, is best done using univariate analysis.

Two areas for improvement of the algorithm as incorporated into the program of Meyer (1989a) were highlighted during the analysis and in the results: parameter limits should be imposed on the original scale (to prevent negative variance component estimates) and occurrence of negative definite (co)variance matrices needs to be avoided (by parameter limitation)

or overcome (possibly by bending, Hayes and Hill, 1980). The algorithm of Thompson and Hill (1990) may not be the best choice for analyses where the common litter of birth variance is small.

Table 4.1: Phenotypic[1] and genetic[2] correlations between performance test traits of Landrace boars.

TMG[3]	Trait	Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
1	Sh Fat	0.22[4]	0.40	0.41	0.41	0.33	0.25	0.25	-0.02
	L Fat	0.44	0.23	0.59	0.59	0.32	0.27	0.27	-0.06
	C Fat	0.37	0.93	0.28	0.95	0.44	0.33	0.33	-0.02
	K Fat	0.40	0.90	1.00	0.30	0.45	0.33	0.33	-0.01
	ADFI	0.20	0.44	0.35	0.41	0.22	0.66	0.66	0.09
	WtG	-0.18	0.23	-0.09	-0.10	0.59	0.20	1.00	-0.68
	ADG	-0.18	0.24	-0.09	-0.10	0.59	1.00	0.20	-0.68
	FCR	0.63	0.09	0.43	0.49	0.14	-0.71	-0.72	0.23
2	Sh Fat	0.24	0.38	0.44	0.44	0.43	0.30	0.30	0.05
	L Fat	0.92	0.12	0.46	0.45	0.31	0.23	0.23	0.02
	C Fat	0.44	0.52	0.36	0.95	0.53	0.42	0.42	0.02
	K Fat	0.33	0.22	1.00	0.31	0.54	0.42	0.42	0.02
	ADFI	0.83	0.42	0.51	0.56	0.19	0.60	0.60	0.24
	WtG	0.23	-0.60	-0.10	-0.04	0.14	0.08	Fail	-0.62
	ADG	0.24	-0.56	-0.19	-0.09	0.07	Fail	0.08	-0.62
	FCR	0.63	0.67	0.66	0.63	0.83	-0.06	-0.14	0.16
3	Sh Fat	0.13	0.37	0.37	0.37	0.39	0.35	0.35	-0.10
	L Fat	-var	0.00	0.49	0.48	0.38	0.36	0.36	-0.13
	C Fat	0.47	-var	0.34	0.96	0.46	0.44	0.44	-0.13
	K Fat	0.57	-var	1.00	0.30	0.48	0.45	0.45	-0.12
	ADFI	1.18	-var	0.07	0.255	0.09	0.76	0.76	0.00
	WtG	0.85	-var	0.37	0.38	0.68	0.17	Fail	-0.64
	ADG	0.83	-var	0.34	0.38	0.67	Fail	0.17	-0.64
	FCR	-0.06	1.00	0.23	0.25	-0.09	-0.88	-0.88	0.18

1 - above diagonal; 2 - below diagonal; 3 - Test Management Group; 4 - univariate estimates of heritability (underlined)

Table 4.2: Phenotypic[1] and genetic[2] correlations between performance test traits of Landrace gilts.

TMG[3]	Trait	Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
1	Sh Fat	0.30[4]	0.38	0.39	0.38	0.12	-0.12	-0.12	0.19
	L Fat	0.68	0.27	0.59	0.59	0.09	-0.29	-0.30	0.36
	C Fat	0.58	0.93	0.39	0.96	0.19	-0.27	-0.28	0.40
	K Fat	0.58	0.94	1.00	0.39	0.19	-0.27	-0.28	0.40
	ADFI	0.39	0.45	0.58	0.58	0.09	0.40	0.39	0.14
	WtG	-0.39	-0.45	-0.38	-0.36	-0.23	0.10	1.00	-0.84
	ADG	-0.41	-0.47	-0.41	-0.38	-0.24	1.00	0.10	-0.84
	FCR	0.41	0.51	0.58	0.55	0.64	-0.91	-0.92	0.16
2	Sh Fat	0.21	0.35	0.34	0.36	0.27	0.31	0.32	-0.19
	L Fat	0.83	0.07	0.42	0.45	0.34	0.30	0.30	-0.09
	C Fat	0.43	0.66	0.25	0.92	0.53	0.53	0.54	-0.23
	K Fat	0.44	0.52	0.99	0.25	0.50	0.52	0.53	-0.24
	ADFI	0.62	-0.12	0.37	0.46	0.17	0.69	0.69	0.01
	WtG	-0.08	-1.00	-0.54	-0.33	0.31	0.19	Fail	-0.72
	ADG	-0.07	-1.00	-0.49	-0.40	0.31	Fail	0.20	-0.73
	FCR	0.76	0.51	0.66	0.65	0.79	-0.24	-0.80	0.26
3	Sh Fat	0.11	0.38	0.34	0.33	0.33	0.21	0.21	-0.02
	L Fat	-0.19	0.03	0.38	0.38	0.30	0.20	0.20	-0.02
	C Fat	0.45	0.96	0.46	0.95	0.35	0.17	0.17	0.05
	K Fat	0.40	0.95	1.00	0.41	0.35	0.18	0.18	0.04
	ADFI	0.62	-0.42	0.05	-0.04	0.05	0.61	0.61	-0.00
	WtG	-0.17	0.11	-0.29	-0.30	0.20	0.15	Fail	-0.79
	ADG	-0.17	0.10	-0.30	-0.31	0.20	Fail	0.15	-0.79
	FCR	0.35	0.40	0.33	0.36	0.35	-0.93	-0.93	0.22

1 - above diagonal; 2 - below diagonal; 3 - Test Management Group; 4 - univariate estimates of heritability (underlined)

Table 4.3: Residual$_1$ and common litter of birth$_2$ correlations between performance test traits of Landrace boars.

TMG$_3$	Trait	Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
1	Sh Fat	0.08$_4$	0.36	0.36	0.37	0.34	0.33	0.33	-0.17
	L Fat	0.84	0.04	0.48	0.49	0.31	0.27	0.27	-0.07
	C Fat	0.90	0.53	0.10	0.92	0.49	0.46	0.46	-0.17
	K Fat	0.88	0.47	0.98	0.07	0.48	0.46	0.46	-0.19
	ADFI	0.56	0.08	0.36	0.31	0.09	0.74	0.74	-0.06
	WtG	0.74	0.55	0.58	0.57	-0.08	0.05	1.00	-0.69
	ADG	0.75	0.55	0.58	0.56	-0.08	1.00	0.05	-0.69
	FCR	-0.21	-0.35	-0.19	-0.21	0.79	-0.71	-0.72	0.18
2	Sh Fat	0.01	0.31	0.36	0.37	0.25	0.28	0.28	-0.20
	L Fat	-1.92	0.00	0.46	0.49	0.31	0.29	0.30	-0.12
	C Fat	1.59	1.24	0.01	0.93	0.50	0.53	0.53	-0.28
	K Fat	1.76	4.44	1.02	0.08	0.47	0.54	0.54	-0.31
	ADFI	1.12	-var	0.99	0.94	0.11	0.70	0.71	-0.01
	WtG	1.81	-var	0.61	0.32	0.39	0.01	Fail	-0.72
	ADG	1.71	-var	0.60	0.34	0.40	Fail	0.01	-0.71
	FCR	1.50	-var	0.78	0.78	0.83	-0.49	-0.47	0.12
3	Sh Fat	0.01	0.43	0.37	0.33	0.27	0.27	0.27	-0.15
	L Fat	-0.25	0.01	0.53	0.52	0.37	0.41	0.41	-0.18
	C Fat	0.48	2.06	0.01	0.94	0.53	0.48	0.48	-0.17
	K Fat	0.77	1.94	1.02	0.01	0.52	0.48	0.48	-0.15
	ADFI	0.97	1.55	1.00	1.01	0.14	0.81	0.81	-0.14
	WtG	-var	1.99	0.41	0.76	-var	0.00	Fail	-0.66
	ADG	-var	1.91	0.99	0.61	-var	Fail	0.00	-0.66
	FCR	0.62	0.36	-0.87	-1.11	1.15	2.48	2.69	0.07

1 - above diagonal; 2 - below diagonal; 3 - Test Management Group; 4 - univariate estimates of common litter of birth effects (underlined)

Table 4.4: Residual$_1$ and common litter of birth$_2$ correlations between performance test traits of Landrace gilts.

TMG$_3$	Trait	Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
1	Sh Fat	0.02$_4$	0.23	0.27	0.25	0.09	-0.03	-0.04	0.11
	L Fat	1.21	0.06	0.40	0.40	-0.00	-0.27	-0.27	0.30
	C Fat	0.95	0.81	0.07	0.92	0.10	-0.28	-0.28	0.36
	K Fat	0.91	0.75	1.00	0.07	0.09	-0.27	-0.27	0.35
	ADFI	-0.55	0.18	0.02	-0.02	0.07	0.45	0.44	0.09
	WtG	-0.67	-0.32	-0.21	-0.31	0.68	0.07	1.00	-0.83
	ADG	-0.59	-0.31	-0.20	-0.30	0.66	1.00	0.08	-0.84
	FCR	0.62	0.72	0.40	0.54	-0.23	-0.86	-0.87	0.04
2	Sh Fat	0.06	0.41	0.44	0.45	0.42	0.30	0.30	0.03
	L Fat	0.04	0.10	0.47	0.46	0.30	0.24	0.24	-0.02
	C Fat	2.25	1.41	0.09	0.95	0.51	0.40	0.40	-0.01
	K Fat	1.73	1.15	1.03	0.09	0.51	0.40	0.40	-0.01
	ADFI	1.56	1.05	1.00	0.82	0.12	0.62	0.62	0.18
	WtG	1.03	0.61	0.85	0.71	0.42	0.08	Fail	-0.65
	ADG	1.03	0.60	0.84	0.69	0.42	Fail	0.09	-0.73
	FCR	0.44	-0.01	0.04	0.05	0.48	-0.64	-0.55	0.04
3	Sh Fat	0.07	0.39	0.32	0.33	0.32	0.26	0.26	-0.09
	L Fat	0.78	0.05	0.35	0.33	0.30	0.19	0.19	-0.04
	C Fat	0.42	0.39	0.02	0.91	0.45	0.31	0.31	-0.05
	K Fat	0.41	0.51	1.00	0.02	0.45	0.32	0.32	-0.07
	ADFI	0.24	0.59	0.97	0.91	0.12	0.64	0.65	-0.04
	WtG	0.34	0.60	0.86	0.79	0.74	0.09	Fail	-0.77
	ADG	0.33	0.60	0.84	0.77	0.75	Fail	0.10	-0.77
	FCR	-0.24	-0.50	-0.40	-0.38	-0.19	-0.76	-0.75	0.06

1 - above diagonal; 2 - below diagonal; 3 - Test Management Group; 4 - univariate estimates of common litter of birth effects (underlined)

Table 4.5: Parameter estimates from bivariate analyses between performance test traits of Landrace boars in TMG 1.

P_1	$Trait_3$	$Trait_2$							
		Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
h^2	Sh Fat		0.227	0.283	0.300	0.222	0.189	0.190	0.215
	L Fat	0.222		0.289	0.293	0.220	0.201	0.200	0.213
	C Fat	0.223	0.226		0.297	0.220	0.205	0.206	0.235
	K Fat	0.223	0.228	0.279		0.221	0.205	0.205	0.237
	ADFI	0.222	0.230	0.283	0.297		0.204	0.204	0.233
	WtG	0.222	0.227	0.283	0.298	0.220		0.201	0.226
	ADG	0.223	0.227	0.282	0.298	0.220	0.201		0.231
	FCR	0.180	0.229	0.279	0.298	0.219	0.207	0.211	
$\bar{h}^2$		0.216	0.228	0.283	0.297	0.220	0.202	0.202	0.227
c^2	Sh Fat		0.038	0.092	0.068	0.089	0.051	0.051	0.184
	L Fat	0.078		0.093	0.073	0.088	0.045	0.046	0.191
	C Fat	0.077	0.035		0.069	0.088	0.050	0.050	0.179
	K Fat	0.077	0.035	0.095		0.088	0.048	0.050	0.178
	ADFI	0.078	0.038	0.093	0.069		0.044	0.044	0.171
	WtG	0.078	0.037	0.093	0.069	0.087		0.045	0.180
	ADG	0.078	0.037	0.092	0.068	0.087	0.045		0.180
	FCR	0.088	0.034	0.090	0.065	0.086	0.053	0.053	
$\bar{c}^2$		0.079	0.036	0.093	0.069	0.088	0.048	0.048	0.180

1 - Parameters: heritability ($h^2 = \sigma_a^2/\sigma_p^2$), common litter of birth effect ($c^2 = \sigma_c^2/\sigma_p^2$) and the average of these across analyses ($\bar{h}^2$ and $\bar{c}^2$);

2 - trait of interest (i.e. all parameters in column labelled 't' relate to trait 't');

3 - 2nd trait in analysis.

Table 4.6: Genetic, residual and phenotypic correlations between performance test traits recorded on Landrace boars in Test Management Groups 2_1 and 3, analysed under model 2.

cor_2	Trait	Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
r_g	Sh Fat	0.23[3]	0.84	0.59	0.57	0.81	0.42	0.43	0.69
		0.13	0.12	0.55	0.39	0.17	-0.50	-0.49	0.66
	L Fat	-var	0.00	0.40	1.00	0.64	0.13	0.11	0.68
	C Fat	0.43	-var	0.33	0.43	0.70	0.17	0.14	0.71
	K Fat	0.46	-var	1.00	0.30	0.36	0.23	0.23	0.83
	ADFI	1.00	-var	0.28	0.36	0.19	0.08	Fail	-0.12
	WtG	0.80	-4.97	0.28	0.28	0.53	0.17	0.07	-0.16
	ADG	0.81	-2.42	0.29	0.31	0.51	1.00	0.17	0.26
	FCR	0.07	-1.05	-0.11	-0.14	0.26	-0.69	-0.68	0.23
r_e	Sh Fat		0.30	0.38	0.39	0.27	0.29	0.29	-0.17
	L Fat	0.46		0.47	0.51	0.34	0.30	0.30	-0.11
	C Fat	0.37	0.56		0.92	0.48	0.53	0.53	-0.30
	K Fat	0.35	0.54	0.94		0.45	0.53	0.54	-0.34
	ADFI	0.27	0.44	0.53	0.53		0.72	0.72	-0.03
	WtG	0.27	0.42	0.50	0.50	0.81		Fail	-0.75
	ADG	0.27	0.42	0.49	0.49	0.81	1.00		-0.74
	FCR	-0.13	-0.16	-0.16	-0.14	-0.06	-0.62	-0.63	
r_p	Sh Fat		0.38	0.44	0.44	0.43	0.31	0.31	0.06
	L Fat	0.36		0.46	0.45	0.30	0.23	0.23	0.02
	C Fat	0.37	0.49		0.95	0.54	0.42	0.42	0.03
	K Fat	0.37	0.48	0.96		0.55	0.42	0.42	0.04
	ADFI	0.40	0.37	0.46	0.48		0.59	0.59	0.26
	WtG	0.36	0.36	0.44	0.44	0.76		Fail	-0.61
	ADG	0.36	0.36	0.44	0.45	0.76	1.00		-0.61
	FCR	-0.09	-0.14	-0.14	-0.14	0.01	-0.63	-0.63	

1 - TMG 2 above diagonal, TMG3 below; 2 - correlation; 3 - univariate h^2

Table 4.7: Approximate standard errors of genetic correlation estimates from Landrace boar Test Management Groups 2 and 3 fitting models 1_1 and 2_2

TMG	$Trait_3$	$Trait_3$							
		Sh Fat	L Fat	C Fat	K Fat	ADFI	WtG	ADG	FCR
2	Sh Fat		0.05	0.06	0.06	0.04	0.22	0.22	0.07
	L Fat	0.02		0.10	0.12	0.17	0.33	0.35	0.12
	C Fat	0.09	0.10		0.00	0.05	0.21	0.22	0.05
	K Fat	0.09	0.13	0.00		0.04	0.20	0.21	0.05
	ADFI	0.05	0.18	0.11	0.10		0.26	0.27	0.04
	WtG	0.20	0.17	0.19	0.18	0.28		ne	0.31
	ADG	0.20	0.19	0.18	0.18	0.29	ne		0.32
	FCR	0.09	0.10	0.07	0.08	0.06	0.25	0.25	
3	Sh Fat		ne	0.15	0.15	-0.00	0.10	0.10	0.25
	L Fat	ne		ne	ne	ne	ne	ne	ne
	C Fat	0.14	ne		0.00	0.11	0.10	0.10	0.10
	K Fat	0.13	ne	0.00		0.11	0.11	0.11	0.11
	ADFI	-0.18	ne	0.18	0.18		0.13	0.14	0.14
	WtG	0.08	ne	0.10	0.10	0.15		0.00	0.08
	ADG	0.09	ne	0.10	0.10	0.15	ne		0.08
	FCR	0.27	ne	0.10	0.11	0.27	0.04	0.04	

1 - below diagonal ; 2 - above diagonal; 3 - correlated traits; ne - not estimable (correlation or standard error of heritability not estimated).

The approximate standard errors were calculated using formulae from Falconer (1981); heritabilities and their standard errors were taken from univariate analyses (chapter 2).

Negative estimates of $se(r_g)$ were due to the estimate of r_g exceeding 1.

CHAPTER 5.

UNIVARIATE GENETIC PARAMETER ESTIMATES FOR REPRODUCTION TRAITS IN PUREBRED LANDRACE AND LARGE WHITE PIGS.

INTRODUCTION

Growth and carcass traits of pigs have been undergoing continuous selection for many generations and it is expected that these traits will reach an optimum point beyond which either the product is unacceptable to the consumer or the fitness of the breeding population can not be maintained. As these limits are approached selection for reproduction traits will become more attractive to breeders looking to maintain genetic and economic improvement in their stock (Hill and Webb, 1982).

Use of BLUP procedures will enable breeders to take account of information from all sources and hence produce good estimates of the predicted breeding values of reproduction traits for animals of both sexes. However, the use of BLUP assumes that variance components for that population are known without error, and so estimates specific to a population are required. Also, in using BLUP it is assumed that the correct model is being fitted to the data.

Maternal effects upon reproduction traits of pigs have been hypothesised for a long time, as combinations of environmental and genetic effects. For example, the size of litter into which the sow was born has been observed as having an influence on the reproductive performance of the sow (Nelson and Robison, 1976; Van der Steen, 1985). Recent results have

indicated the presence of a maternal genetic effect for reproduction traits (Southwood and Kennedy, 1989), and this may bias heritability estimates if omitted from the model fitted.

Haley *et al.* (1988) reviewed parameter estimates for litter size, and concluded that the heritability of litter size born alive was around 0.09 with a repeatability of 0.15. Since then Animal Model REML estimates of the heritability of reproduction traits across parities have been produced. Gu *et al.* (1989b) found that the heritability and repeatability of reproduction traits were around 0.11 and 0.15, respectively, in Landrace and Large White based selection lines, and Sorensen (1990) gave values of 0.12 and 0.19 for the heritability and repeatability of number born alive in a Yorkshire population.

Analysing gilt data from purebred and crossbred Yorkshire and Landrace gilts, Southwood and Kennedy (1990) produced heritability estimates of between 0.06 and 0.13 (across data sets and traits) fitting a model which included a maternal genetic effect.

By use of Individual Animal Model REML, the maternal genetic and common litter of birth effects can be assessed for our population and parameter estimates obtained. The populations of animals in this study are closed commercial Landrace and Large White lines, undergoing selection for growth and carcass traits, with a high management input via crossfostering, early weaning and extra suckling which may affect the maternal and common environmental variances. The parameter estimates will therefore help to give some indication of the importance of the maternal genetic and common litter of birth effects in these nucleus populations.

MATERIAL AND METHODS

Reproduction records were collected on 1891 Landrace and 2432 Large White sows (5291 and 7683 records, respectively) all of which were born and performance tested in the same purebreeding nucleus herd, as described in Chapter 2 (all sows in this analysis had performance test records included in previous analyses). Selection was carried out in the nucleus herd upon an index of performance test traits. Depending on their selection index value, gilts went on to breed in the nucleus herd or one of 4 multiplier herds (not the same herds for both breeds) in this pyramid or not at all. Selection was not widely practiced on reproduction traits. A few animals in the data had prolonged reproductive lives and possibly promotion from multiplier to nucleus herd level as a result of having a high average number of offspring born over their first three parities.

Total number born (NB) including stillborn piglets, number born alive (NBA), total litter weight (LWt; recorded in kg, including weight of stillborn piglets), average piglet weight (PWt = LWt/NB; kg/piglet) and gestation length (GL; days) were analysed in this study. These five traits were selected as representative of the sow's reproductive performance after preliminary Least-Squares analysis showed that they were highly correlated with their constituent traits (numbers of boars or gilts born and stillborn) and other possible traits, such as length of suckling period, had very low (often negative) genetic variance estimates. Concerns over non-Normality of the data and confounding with management effects also influenced the rejection of weaning to service intervals, length of suckling period, number of mummified piglets produced and farrowing interval.

Although records were made of a number of variables related to litter

performance to weaning (number of offspring weaned, weight of offspring weaned) these were discarded because they were expected to be severely confounded with management practices of which no records exist. These practices include cross-fostering shortly after farrowing (giving rise to artificially increased or decreased numbers in a litter), early weaning of the most successful members of a litter and extended suckling periods for good dams (extra piglets may be placed with a sow after her litter has been weaned).

Table 5.1: Number of Landrace and Large White sows and records in each herd.

Herd	Key	Landrace			Large White		
		$n_{sow/H}$	$n_{rall/H}$	$n_{r1/H}$	$n_{sow/H}$	$n_{rall/H}$	$n_{r1/H}$
nucleus	Δ	478	1235	379	526	1270	413
multi.1	◆	478	1366	364	94	206	84
multi.2	x	254	717	172	366	1336	296
multi.3	◇	638	1904	458	-	-	-
multi.4	■	43	69	43	-	-	-
multi.5	o	-	-	-	717	2620	544
multi.6	+	-	-	-	729	2251	622

Where: multi.i - is multiplier herd i;

key - are symbols representing that herd in figures 5.1 - 5.8

$n_{sow/H}$ - is the number of sows with records in that herd;

$n_{rall/H}$ - is the number of litter records in that herd;

$n_{r1/H}$ - is the number of first parity litter records in that herd.

The recording system used by the company allowed storage of up to 5 records per sow, which meant that sows which reached parity 6 or greater

had earlier parity records overwritten (i.e. sows which reached parities 6, 7 and 10 would be expected to have records on parities 2...6, 3...7 and 6...10 respectively). Because of this the Landrace data contain up to five records for a sow which do not necessarily come from the first five parities and need not be of consecutive parities (due to missing records). However, the Large White data contains some sows with more than five records available because of retrieval of additional archived data by the breeding company.

Table 5.2: Distribution of Landrace and Large White reproduction records across parities.

Parity	Landrace	Large White
1	1416	1959
2	1185	1643
3	919	1356
4	657	996
5	457	704
6	304	463
7	180	298
8	96	163
9	45	76
10	21	22
11	11	3

Where each cell contains the number of litter records for sows of that parity.

Tables 5.1 and 5.2 show the distribution of sows and records across herds and parities, respectively, and from these and table 5.3 it can be seen that the data on the two breeds are similarly structured.

Data validation:

Pedigree information and dates of birth were checked in unison with the sow's performance test record (see Chapter 2). At the same time, offspring records in the performance test data were matched to the reproduction record of the dam and the dates associated with those records were checked for correspondence.

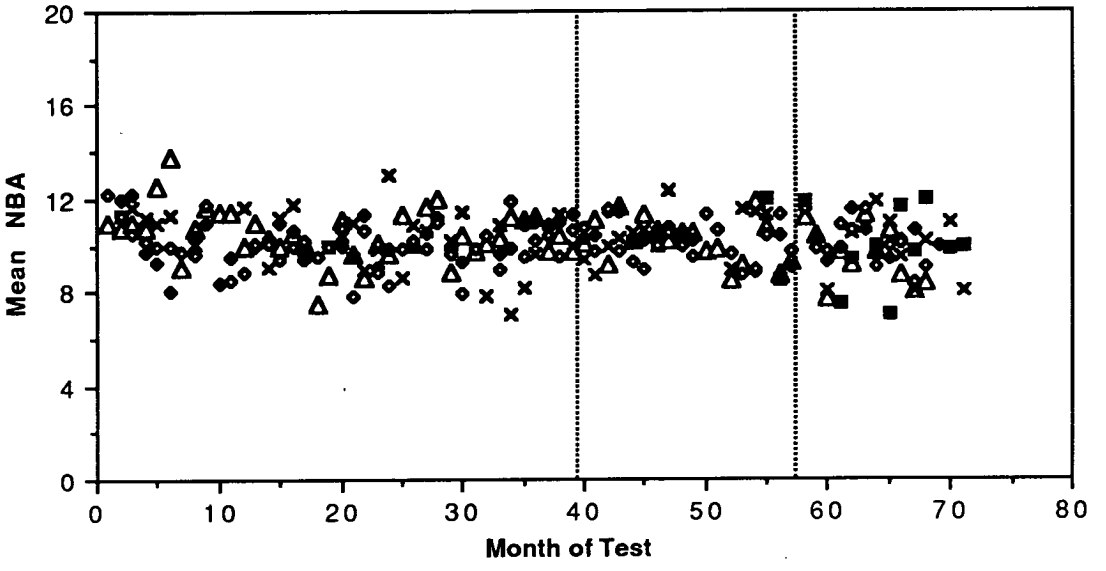
Records with errors for a particular trait after all pedigree and date records had been validated could only be identified if they resulted in extreme values for that trait. Records which were identified as containing data errors of this type were deleted from the data set.

Data sets analysed:

The two breeds were analysed separately, there being no links between the two lines. Within each breed, data sets were constructed to allow both the analysis of traits across all parities and in the first parity only.

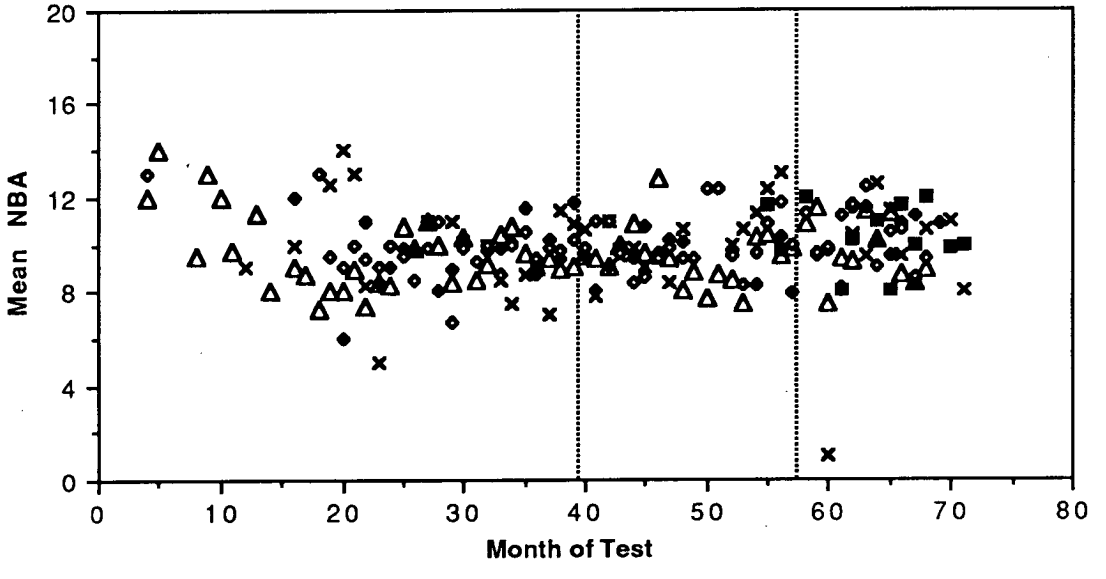
In the analysis of the performance test traits, data were subdivided into Test Management Groups (see Chapter 2) based upon changes in the performance test regime (feeding scales, length of time spent on test and fixed weight or time period test) and the health status of the herd. Figures 5.1 to 5.8 show phenotypic means of number born alive and average piglet weight for first parity and all parity data sets against the month in which the sow completed performance test (month of test is defined as constituting four consecutive test batches, where all animals finishing test in the same week were assigned to the same test batch). In this way, the Test Management Groups identified in the perfomance test data can be related to the reproduction data.

Figure 5.1: Graph of phenotypic mean of number born alive against month of test of sow for Landrace parity records on all parities.



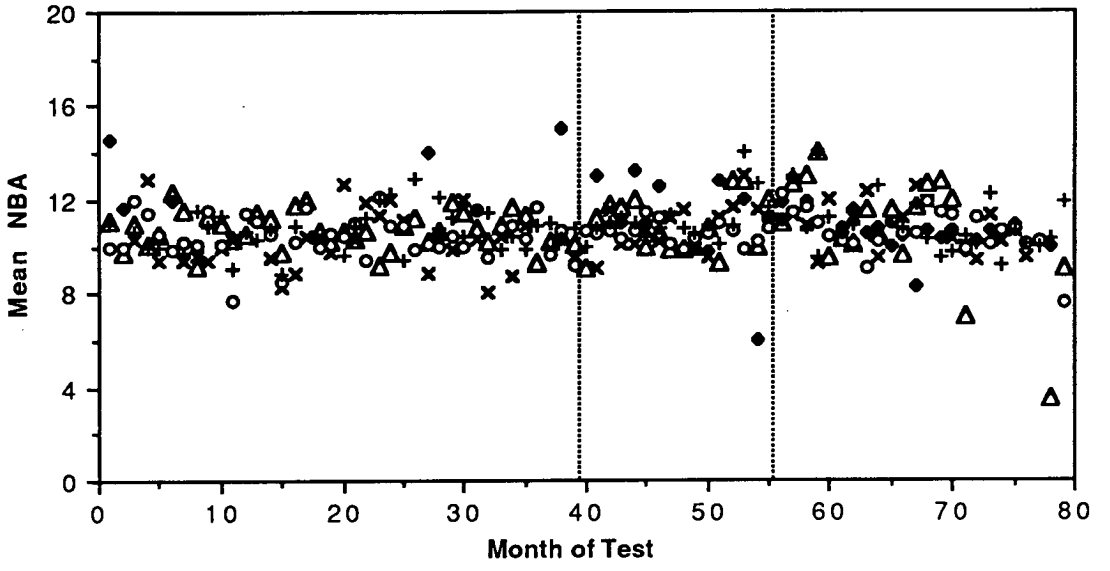
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.2: Graph of phenotypic mean of number born alive against month of test of sow for Landrace records from the first parity.



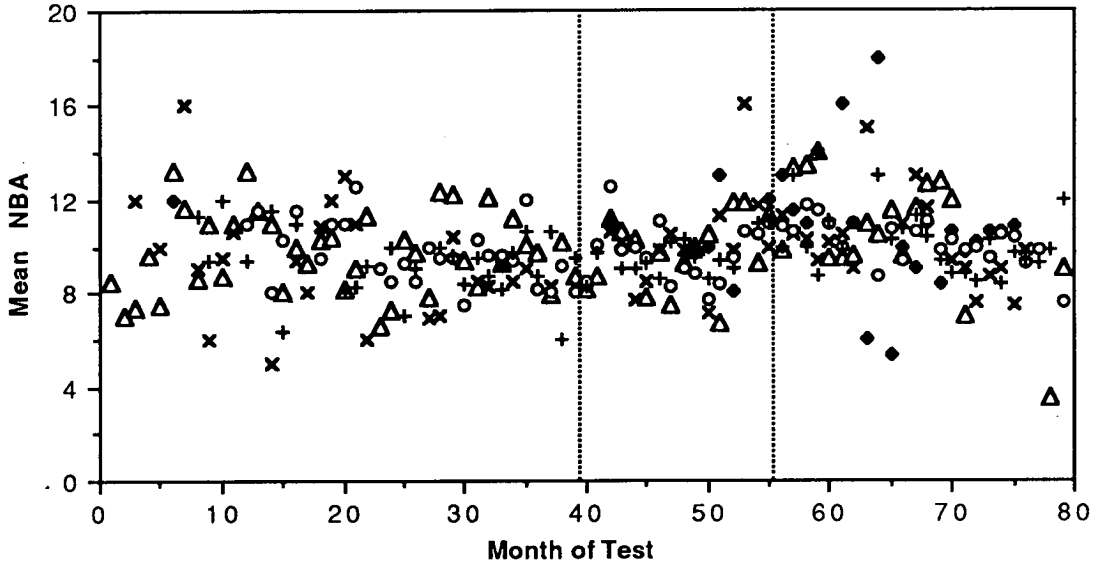
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.3: Graph of phenotypic mean of number born alive against month of test of sow for Large White records on all parities.



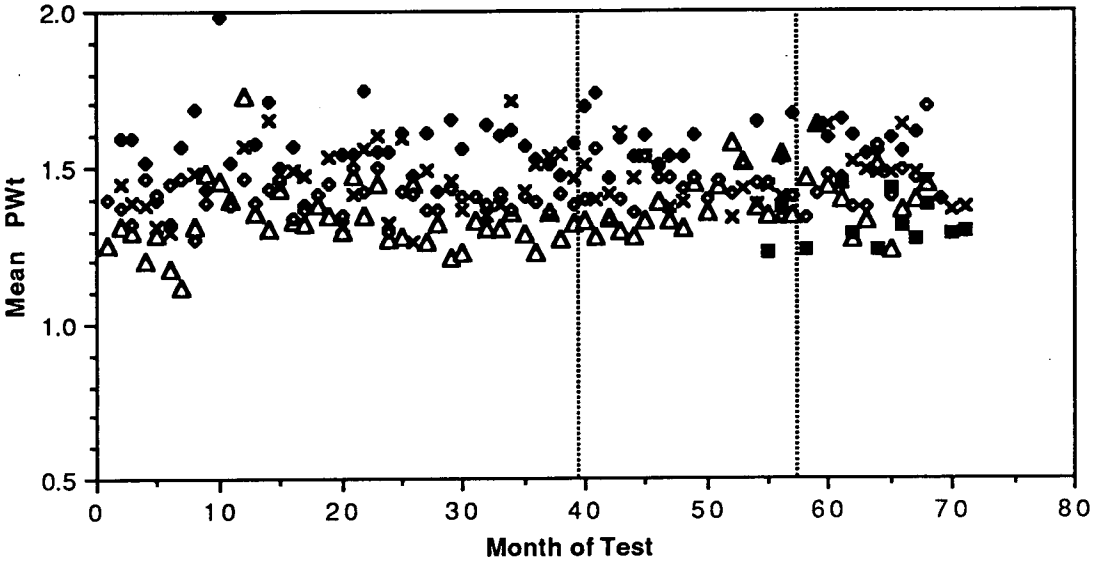
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.4: Graph of phenotypic mean of number born alive against month of test of sow for Large White records from the first parity.



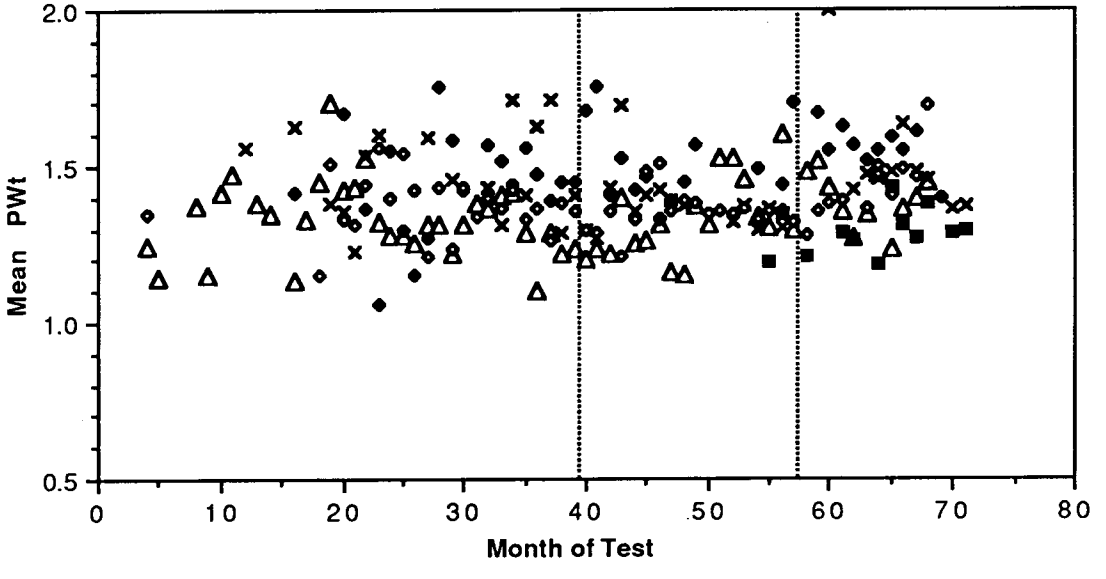
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.5: Graph of phenotypic mean of average piglet weight against month of test of sow for Landrace records on all parities.



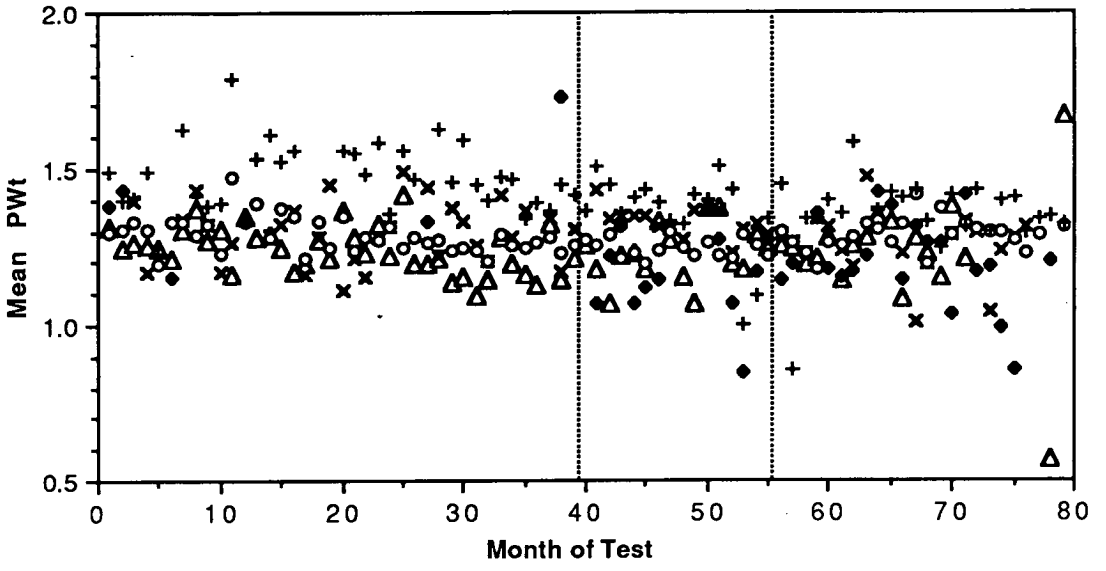
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.6: Graph of phenotypic mean of average piglet weight against month of test of sow for Landrace records from the first parity.



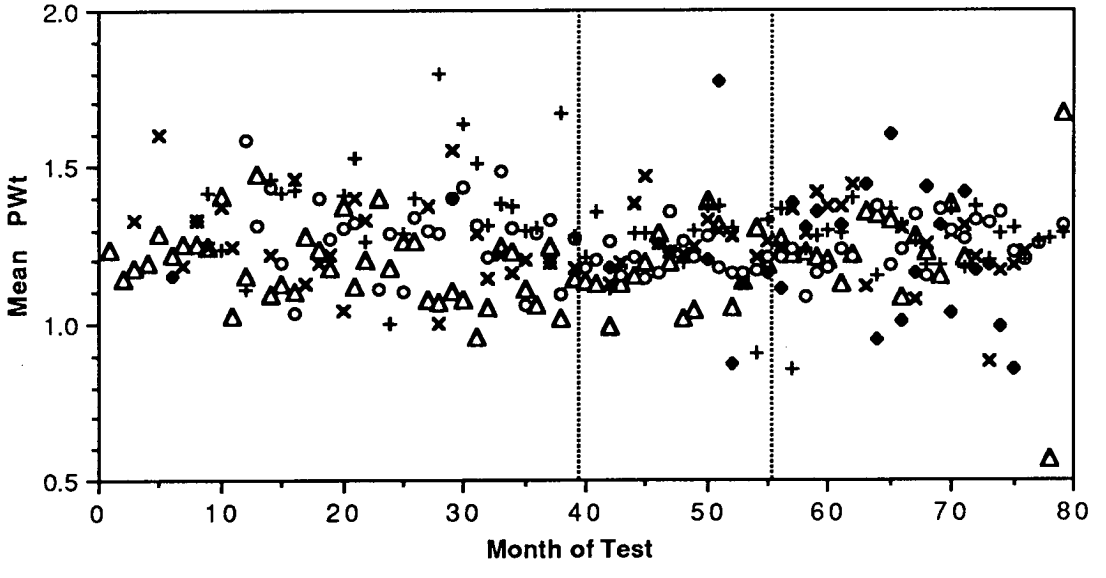
Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.7: Graph of phenotypic mean of average piglet weight against month of test of sow for Large White records on all parities.



Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

Figure 5.8: Graph of phenotypic mean of average piglet weight against month of test of sow for Large White records from the first parity.



Where:- symbols are as defined in table 5.1 and vertical lines delimit Test Management Groups as defined in chapter 2.

From figures 5.1-5.8 it can be seen that there is no discernible difference between mean performance of gilts and sows for either NBA or PWt related to the gilt's Test Management Group, regardless of the herd in which her reproduction records were collected. The means for the earliest and latest Test Management Groups have large standard errors, since there are few records involved in these estimates; also the standard errors of the first parity means are higher than those of the all parity estimates.

The differences between the Large White and Landrace data sets, caused by continued collection of Large White data whilst performance test and reproduction data from the Landrace breed was being analysed and retrieval of archived Large White data, are evident from the graphs; early and late Test Management Groups are represented by more records (in particular, more first parity records exist for Large Whites tested at the beginning of the data collection period than for Landrace contemporaries, for which they may have been overwritten by later parity records), and there are more reproduction records from females which were performance tested later in the final Test Management Group.

Since there was no phenotypic effect of the Test Management Group of the sow observed upon the data (figures 5.1-5.8), no account was taken of these changes in the following analyses. The inclusion of farrowing period nested within herd (FHP, see table 5.3) as a fixed effect should take some account of any differences caused by health status or test management regime, and if the genetic correlation between perfomance test traits and reproduction traits is low, as is indicated by the results reviewed by Haley *et al.* (1988), then these effects should be small.

Table 5.3: Summary of population structure for sows of both breeds.

Breed	Parities	n_{rec}	n_{sow}	n_{anim}	n_{SBP}	n_{FHP}	n_s	n_d
LR	1-11	5291	1891	2202	36	123	88	552
	1	1416	1416	1753	33	116	81	450
LW	1-11	7683	2432	2856	40	179	117	722
	1	1959	1959	2430	40	164	110	631

Where: - breed and parity are used to identify data sets;

n_{rec} - number of records in data set;

n_{sow} - number of sows with records in data set;

n_{anim} - number of animals in analysis (including those in pedigree only);

n_{SBP} - number of eight week periods in which sows were born;

n_{FHP} - number of eight week farrowing period-farrowing herd combinations;

n_s - number of sires of sows;

n_d - number of dams of sows.

Analyses performed:

Each data set was analysed using the univariate Derivative-Free REML (DFREML) algorithm developed by Dr. K. Meyer. This algorithm is an extension of the method proposed by Smith and Graser (1986, and Graser *et al.*,1987) to allow simultaneous estimation of multiple random effects (Meyer,1988 and 1989a).

Making use of this capability it was possible to estimate a common environmental effect, which would be expected to affect littermates, or more precisely litter of birth mates since cross-fostering was widely practiced in the population, and an effect of the maternal genotype, as in Chapter 2.

For analyses of traits across all available parities, a permanent environmental effect of the sow was included in the analysis. This assumes that the traits are under the same genetic control in each parity, which implies a genetic correlation of one between parities. Estimates of these between parity correlations are reviewed by Haley *et al.* (1988), and it was concluded that there was no evidence for treating the traits in different parities as separate traits.

Fixed effect assessment:

Preliminary Least-Squares analysis of the data using LSML76 (Harvey, 1976) indicated that there were significant effects on all traits associated with parity of the sow, period in which the sow was born, period in which sow farrowed and unit on which the sow farrowed, whilst no significant effect was found to be associated with size of the litter into which the sow was born and the sow's performance test record. These results are in line with those of Gu *et al.* (1989b).

The hypothesis that farrowing period should be regarded as being nested within farrowing unit, since it could not be assumed that the management and environmental conditions would be identical across herds during a given time period, was thus investigated.

Landrace reproduction data from all parities were analysed using DFREML fitting five different fixed effect models, and the parameter estimates were compared subjectively. The use of an F test to compare residual variances was rejected because the large number of degrees of freedom associated with each estimate caused any difference to be highly significant. A likelihood ratio test is inappropriate for assessing fixed effect

models using REML. The models fitted include the same random effects - an additive genetic effect, a permanent environmental effect associated with the sow and a residual effect - and were:

$$Y_{ijklm} = a_i + s_i + P_j + SBP_k + AFE_{lm} + e_{ijklm} \quad (1)$$

Where:

Y_{ijkl} - is the phenotypic record of sow i for the trait;

a_i - is the random additive genetic effect of the ith sow;

s_i - is the permanent environmental effect of the ith sow;

P_j - is the fixed effect of the jth parity;

SBP_k - is the fixed effect of the kth sow birth period (eight week periods);

AFE_{lm} - is the lth combination of fixed effects associated with that sow for model m. The four different models used were:

Farrowing unit (FU) for m = 1,

Farrowing period (FP) for m = 2,

FU and FP for m = 3,

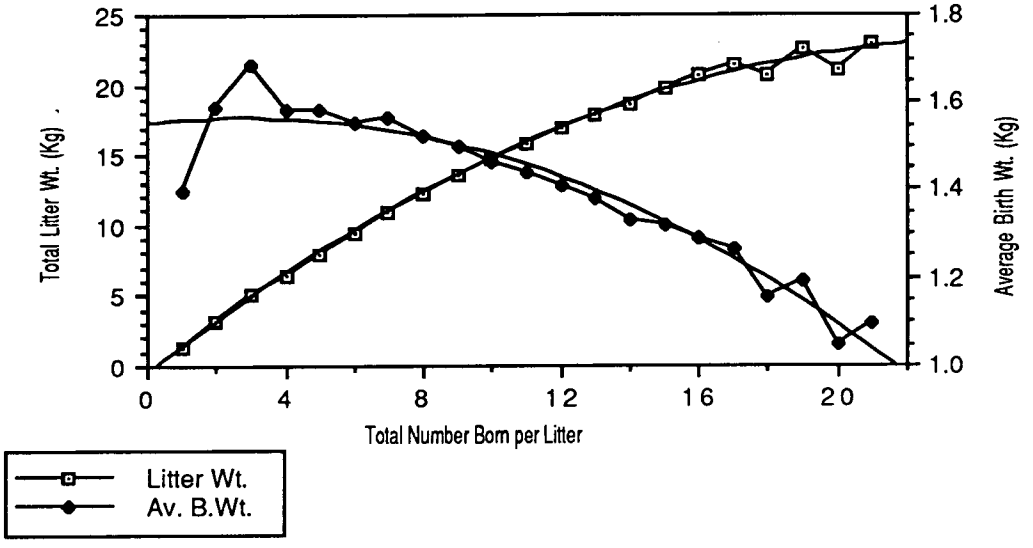
FP within FU for m = 4;

e_{ijklm} - is the random environmental effect associated with the record, Y_{ijklm}.

Correction of PWt and LWt for total number born:

There is a relationship between the total litter weight, average piglet weight and total number born. It may be desirable to select for either of these weight traits adjusted for the total number born, and parameters were estimated including total number born as a covariate in the analysis.

Figure 5.9: Graph of total litter weight and average piglet weight against total number born for Landrace all parity data.



Landrace average piglet weight and litter weight data from all parities was analysed using DFREML, fitting linear and quadratic relationships with the covariate total number born. The choice of linear and quadratic regressions was made on the basis of preliminary phenotypic linear regression analysis of the data. The graph in figure 5.9 shows mean PWt and mean LWt for each value of NB plotted against NB. Although there appears to be an increase in the mean PWt with total number born at low values of NB, there were few records for each of these points and hence they have large standard errors.

Only all parity data were used because there appeared to be a similar relationship between the traits in the first parity, and hence only one analysis was considered necessary to assess the different models. Other fixed effects included in the model were parity of sow at this farrowing, birth

period of sow and farrowing period nested within farrowing herd of sow.

Comparison of different random effect models:

The effect of including or omitting the random common litter of birth effect and the additive maternal genetic effect (correlated with the additive direct genetic effect) were studied by a sequence of analyses of the Landrace data for first and all parities under different random effect models.

The algorithm of Meyer (1989a) allows the fitting of a maximum of three random effects, excluding the residual effect. Because of this and the need to fit a permanent environmental effect associated with the sow to allow estimation of the repeatability, the common litter of birth and maternal genetic effects could not be fitted simultaneously to the all parity data. In this way, the number of parameters in the model and hence the dimensionality of the search procedure and the time taken to perform it were reduced.

The linear model in matrix notation is:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e} \qquad (2)$$

Where:

- **Y** - is a vector of n_{rec} observations;
- **b** - is a vector of n_f fixed effects;
- **X** - is an n_{rec} x n_f incidence matrix relating fixed effects to records;
- **u** - is a vector of n_u random effects;
- **Z** - is an n_{rec} x n_u incidence matrix relating random effects to records.

The following variance structure was assumed throughout:

$$V(\mathbf{u}) = \mathbf{G};$$

$$V(\mathbf{e}) = \mathbf{R} = \sigma_e^2\mathbf{I};$$

$$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$$

$$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}.$$

The differences between the models fitted can then be illustrated in terms of the changes made to the random effects vector, **u**, and to the assumed variance-covariance matrix, **G**, which are summarised in Tables 5.4 and 5.5 for all parity and first parity analyses, respectively.

The incidence matrix, **Z**, changed to accomodate changes in the vector, **u**, becoming block-diagonal when more than one random effect was included, each block consisting of the incidence matrix for one of the random effects. The fixed effect vector, **b**, contains sow's birth period, farrowing period nested within farrowing unit, and for analyses of all parity data, parity. The vector, **b**, and its incidence matrix, **X**, remain unaltered across analyses comparing random effects.

The dimensions of the random effect vector, **u**, and the incidence matrix, **Z**, are dependent upon the number of random effects included in the model and the length of the random effect vector for each one. For Landrace sows the number of levels of the common litter of birth effects were 1095 and 834 for all parity and first parity analyses, respectively.

Table 5.4: Summary of differences between models fitted for analysis of all parity data.

Model:	1_{All}	2_{All}	3_{All}
Parameters:	h^2, s^2	h^2, s^2, c^2	h^2, m^2, σ_{am}/σ_p^2, s^2
Variance components:	σ_a^2, σ_s^2, σ_e^2	σ_a^2, σ_s^2, σ_c^2, σ_e^2	σ_a^2, σ_m^2, σ_{am}, σ_s^2, σ_e^2
u'	**(a' s')**	**(a' s' c')**	**(a' m' s')**
G	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \sigma_s^2\mathbf{I} \end{bmatrix}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \sigma_s^2\mathbf{I} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \sigma_{am}\mathbf{A} & \mathbf{0} \\ \sigma_{am}\mathbf{A} & \sigma_m^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \sigma_s^2\mathbf{I} \end{bmatrix}$

Where:

A - is the numerator relationship matrix;

a - is a vector of n_{anim} direct additive genetic effects, $\mathbf{a} \sim N(0, \sigma_a^2\mathbf{A})$;

m - is a vector of n_{anim} maternal additive genetic effects, $\mathbf{m} \sim N(0, \sigma_m^2\mathbf{A})$;

s - is a vector of n_{sow} permanent environment effects, $\mathbf{s} \sim N(0, \sigma_s^2\mathbf{I})$;

c - is a vector of n_c common environmental (litter of birth) effects, $\mathbf{c} \sim N(0, \sigma_c^2\mathbf{I})$;

h^2 - is the heritability, σ_a^2 / σ_p^2;

m^2 = σ_m^2 / σ_p^2;

s^2 = σ_s^2 / σ_p^2;

c^2 = σ_c^2 / σ_p^2;

$\sigma_{am}\mathbf{A}$ - is the covariance between **a** and **m'**.

Table 5.5: Summary of differences between models fitted for analysis of first parity data.

Model:	1_{1st}	2_{1st}	3_{1st}
Parameters:	h^2	h^2, c^2	h^2, m^2, σ_{am}/σ_p^2, c^2
Variance components:	σ_a^2, σ_e^2	σ_a^2, σ_c^2, σ_e^2	σ_a^2, σ_m^2, σ_{am}, σ_c^2, σ_e^2
u'	**a'**	**(a' c')**	**(a' m' c')**
G	$\sigma_a^2\mathbf{A}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$	$\begin{bmatrix} \sigma_a^2\mathbf{A} & \sigma_{am}\mathbf{A} & \mathbf{0} \\ \sigma_{am}\mathbf{A} & \sigma_m^2\mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \sigma_c^2\mathbf{I} \end{bmatrix}$

Where all definitions are as defined for table 5.4.

RESULTS

Phenotypic means of the traits are given in table 5.6. Results from the analyses presented are the estimates of the (co)variance components as a proportion of the total phenotypic variance, and the residual and phenotypic variances. For analyses on data on all parities the repeatability, r, is given as the sum of the variance components due to genetic and permanent environmental effects as a proportion of the phenotypic variance (Falconer,1981).

Table 5.6: Phenotypic means of reproduction traits of Landrace and Large White sows.

Trait	Units	Landrace:		Large White:	
		All Parities	First Parity	All Parities	First Parity
NB	pigs/litter	10.6	9.91	11.4	10.3
NBA	pigs/litter	10.2	9.50	10.6	9.80
LWt	kg/litter	15.1	13.6	14.6	12.6
PWt	kg/pig	1.44	1.40	1.31	1.25
GL	days	115	115	115	115

Landrace data

Fixed effect assessment:

Table 5.7 contains the results from analyses of Landrace all parity data fitting different combinations of farrowing herd and farrowing period as fixed effects alongside the parity and sow's birth period effects.

Estimates of heritabilities are consistent across models and traits. However, for model 2 (farrowing unit omitted from the model) the estimates of s^2 increase for LWt, PWt and GL, and this inflates the estimates of

Table 5.7: Results of analysis of all parity reproduction data from the Landrace breed under different fixed effect models.

Trait:	model*1	σ_e^2	σ_p^2	h^2	s^2	r
NB	1	6.58	8.21	0.11	0.09	0.20
	2	6.56	8.22	0.12	0.09	0.20
	3	6.57	8.20	0.11	0.08	0.20
	4	6.54	8.16	0.12	0.08	0.20
NBA	1	6.34	7.83	0.10	0.09	0.19
	2	6.34	7.85	0.10	0.09	0.19
	3	6.36	7.83	0.10	0.09	0.19
	4	6.32	7.79	0.10	0.09	0.19
LWt	1	12.1	15.5	0.13	0.09	0.22
	2	12.0	16.2	0.14	0.12	0.26
	3	12.0	15.4	0.14	0.08	0.22
	4	11.9	15.3	0.15	0.08	0.22
PWt	1	0.0389	0.0534	0.20	0.07	0.27
	2	0.0382	0.0602	0.20	0.16	0.37
	3	0.0384	0.0530	0.21	0.07	0.28
	4	0.0373	0.0521	0.21	0.07	0.28
GL	1	1.44	2.01	0.19	0.09	0.28
	2	1.43	2.17	0.19	0.16	0.34
	3	1.42	1.98	0.19	0.09	0.28
	4	1.36	1.90	0.20	0.09	0.29

*1 1 - Farrowing unit (FU) fitted; 2 - Farrowing period (FP) fitted; 3 - FU and FP fitted; 4 - FP nested within FU fitted.

phenotypic variance and the repeatability, for there to be no reduction in h^2 the estimate of the additive genetic variance must also have increased . Residual variance estimates are similar across models and traits, but the lowest estimate is always associated with model 4 (farrowing period nested within herd), with the associated decrease in phenotypic variance resulting in slight increases in parameter estimates.

Correction of PWt and LWt for total number born:

Table 5.8 contains results of analyses of Landrace all parity data with additive genetic and permanent environmental random effects fitted and the same fixed effect structure as model 4 in the fixed effect assessment. Differences between the models are due to inclusion of total number born as a covariate, with either linear or linear plus quadratic regression terms fitted.

Table 5.8: Results of comparison of different models for Litter Weight and Average Piglet Weight - linear and quadratic corrections for total number born in the litter.

Trait:	Correction:	σ_e^2	σ_p^2	h^2	s^2	r
PWt	None	0.0373	0.0521	0.22	0.07	0.28
	Linear	0.0316	0.0452	0.24	0.07	0.30
	Quadratic	0.0317	0.0452	0.23	0.06	0.30
LWt	None	11.885	15.317	0.15	0.08	0.22
	Linear	3.225	4.590	0.21	0.08	0.30
	Quadratic	3.119	4.484	0.24	0.07	0.30

It can be seen that linear correction of PWt and LWt for total number born increases the heritability estimates, decreasing the residual variances.

Inclusion of a quadratic term has little effect on the estimation of parameters for PWt, but causes a further decline in the residual variance and an increase in the h^2 estimate for LWt.

Comparison of different random effect models:

Table 5.9 contains the results of analyses of Landrace reproduction data from across all parities and the first parity individually under models including different random effects as described in tables 5.4 and 5.5 and acompanying text. The fixed effect models included 8 week sow birth period, 8 week farrowing period nested within farrowing unit and parity (for analysis of data on all parities).

No likelihood ratio test statistics are included in the tables, with one exception none were significant ($p>0.1$). The exception was the estimate of the common litter of birth effect ($c^2=0.02$) across all parities for NBA.

Large White data

Table 5.10 contains the results of analyses of Large White reproduction data from the first parity and across all parities, fitting the same model as in fixed effect assessment model 4. On the basis of the Landrace results it was assumed that maternal genetic effects and common litter of birth effects were negligible and these were omitted from the analyses.

Table 5.9: Results of analysis of Landrace reproduction data under models containing different random effects.

Trait:	M*1	σ^2_e	σ^2_p	h^2	c^2	m^2	σ_{am}*2	r_{am}	s^2	r
NB	1_{All}	6.54	8.16	0.12					0.08	0.20
	2_{All}	6.54	8.16	0.12	0.03				0.06	0.20
	3_{All}	6.55	8.13	0.10		0.02	-0.00	-0.06	0.08	0.20
	1_{1st}	7.22	8.07	0.11						
	2_{1st}	7.21	8.08	0.11	0.00					
	3_{1st}	7.07	7.71	0.15	0.01	0.01	-0.05	-1.00		
NBA	1_{All}	6.32	7.79	0.10					0.09	0.19
	2_{All}	6.33	7.78	0.09	0.02				0.08	0.19
	3_{All}	6.32	7.76	0.09		0.01	-0.00	-0.09	0.09	0.19
	1_{1st}	7.03	7.89	0.11						
	2_{1st}	7.02	7.90	0.11	0.00					
	3_{1st}	7.02	7.89	0.11	0.00	0.00	-0.00	-0.03		
LWt	1_{All}	11.9	15.3	0.15					0.08	0.22
	2_{All}	11.9	15.3	0.13	0.02				0.07	0.22
	3_{All}	11.9	15.3	0.13		0.01	0.00	0.03	0.08	0.22
	1_{1st}	13.0	14.7	0.11						
	2_{1st}	13.1	14.7	0.11	0.00					
	3_{1st}	13.0	14.7	0.11	0.00	0.00	-0.00	-0.01		
PWt	1_{All}	0.0373	0.0521	0.21					0.07	0.28
	2_{All}	0.0373	0.0521	0.21	0.00				0.07	0.28
	3_{All}	0.0373	0.0522	0.21		0.00	0.00	0.40	0.07	0.28
	1_{1st}	0.0397	0.0508	0.22						
	2_{1st}	0.0383	0.0508	0.20	0.05					
	3_{1st}	0.0378	0.0505	0.22	0.05	0.00	-0.01	-0.98		
GL	1_{All}	1.35	1.90	0.20					0.09	0.29
	2_{All}	1.34	1.90	0.20	0.00				0.09	0.29
	3_{All}	1.34	1.90	0.20		0.01	0.00	0.01	0.09	0.29
	1_{1st}	1.89	2.42	0.22						
	2_{1st}	1.90	2.42	0.22	0.00					
	3_{1st}	1.87	2.36	0.25	0.00	0.01	-0.03	-0.61		

*1 - random effect model as defined in tables 5.4 and 5.5; *2 - as a proportion of σ^2_p.

Table 5.10: Results of univariate DFREML analyses of reproduction traits of Large White sows

Trait	Parities	σ_e^2	σ_p^2	h^2	s^2	r
NB	All	7.66	10.0	0.15	0.08	0.23
	First	7.23	9.42	0.23		
NBA	All	7.48	9.42	0.11	0.09	0.20
	First	7.45	9.31	0.20		
LWt	All	11.1	14.8	0.14	0.11	0.25
	First	10.6	12.7	0.17		
cLWt	All	3.42	4.81	0.17	0.12	0.29
	First	3.11	3.81	0.19		
PWt	All	0.0349	0.0503	0.19	0.12	0.31
	First	0.0381	0.0482	0.21		
cPWt	All	0.0284	0.0416	0.19	0.12	0.31
	First	0.0335	0.0406	0.18		
GL	All	1.11	1.45	0.14	0.10	0.24
	First	1.70	1.85	0.08		

Where cLWt and cPWt are LWt and PWt analysed with NB fitted as a linear regression coefficient.

DISCUSSION

Fixed effect assessment:

Estimates of the heritability were consistent across different fixed effect models for all of the traits studied. Models 2 and 4 (farrowing unit omitted from the model and farrowing period nested within farrowing unit respectively) both produced noticeable changes in variance component estimates for some or all of the traits, however.

Omission of the farrowing unit from the model causes an increase in the permanent environmental variances of LWt, PWt and GL. Because all records on a Landrace sow are from the same unit, the farrowing unit will be a permanent effect on the sow. As a result, the between farrowing unit variance is included in the permanent environmental variance. It can be concluded from the observed increase in the permanent environmental variance for LWt, PWt and GL that the farrowing unit has a larger effect on LWt, PWt and GL than on NB or NBA.

Following the same argument, when the farrowing period is left out of the model the between farrowing period variance would be expected to form part of the residual variance estimate, because all litter records on a sow were associated with a different farrowing period level. In line with this hypothesis, slightly higher estimates of σ_e^2 were observed for model 1 for NB, LWt, PWt and GL, although the magnitude of these increases were low.

Estimates of σ_e^2 for all traits under model 4 were marginally lower than those from analyses under all other fixed effect models. This implies that there is a small, but observable, interaction between farrowing period and farrowing unit. That is, there is some difference between farrowing in period

t on unit i and in period t on unit j, which may be due to changes in sow, and in particular farrowing, management, feeding, housing or health status.

Correction of PWt and LWt for total number born:

By definition, total litter weight at birth can be expressed as the product of the average piglet weight and the total number born. From this the correlation between PWt and NB can be approximated using the formula for the variance of the product of two variables to estimate the covariance between PWt and NB for any random effect. The covariance is approximated by:

$$cov(PWt,NB) = \{var(LWt) - \bar{x}^2_{PWt}.var(NB) - \bar{x}^2_{NB}.var(PWt)\} / 2\bar{x}_{PWt}\bar{x}_{NB}$$

Estimates of the variances and phenotypic means required for this procedure were produced by the univariate analyses. The following estimates of genetic and residual correlations between uncorrected PWt and total number born were produced:

	Landrace		Large White	
Parities:	1-11	1	1-11	1
r_g	-0.31	-0.44	-0.49	-0.60
r_e	-0.39	-0.34	-0.42	-0.35

From these estimates it can be seen that there were genetic and environmental relationships between total number born and average piglet weight.

The DFREML analyses fitting linear and linear plus quadratic regressions of NB on PWt indicate that the linear model removes most of the variation in average piglet weight due to NB.

In figure 5.9, total litter weight increased with NB, however since each piglet gets progressively smaller this relationship was asymptotic. In accordance with this, a quadratic regression of LWt on NB has a noticeable effect above that of a linear correction.

Comparison of different random effect models:

Estimates of common litter of birth effects and maternal genetic effects were found to be very low in both first parity and all parity analyses in Landrace data.

The estimates of common litter of birth effects were generally lower than those found for performance test traits in the same population (which were generally around 0.05; see chapter 2). This may be because the female is further removed from the common environment when she farrows, and so other environmental factors will be accumulating which mask this effect, or the effect is not permanent.

Maternal genetic effects are considerably lower than those reported by Southwood and Kennedy (1990) for Canadian Landrace gilts (m^2 estimates for NB and NBA of 0.05 and 0.08, respectively).

The small size of the maternal genetic effects may be explained by the large amount of cross-fostering practiced, often within 24 hours of birth, which means that sows who are littermates at birth do not necessarily share the same post-natal environment. If so, this implies that any maternal genetic effect acts post-natally, which is also reported by Southwood and Kennedy (1990) who found that estimates of m^2 for number weaned were generally higher than for NB and NBA. Therefore, *in utero* or cytoplasmic effects could not be major contributors to estimates of c^2 and m^2, as these

would be observed whether or not cross-fostering was practiced.

Common family environmental effects and maternal genetic effects were lower in gilt litters than across all parities, when one might expect to see a greater effect in the first litter, subsequently decreasing with time. Although sampling error cannot be discarded as the possible cause of this, it could also be due to confounding of c^2 and m^2 estimates across all parities as these effects were not estimated simultaneously, or better estimation across all parities because there is more information available on each dam or litter of birth.

Correlations between additive direct and additive maternal genetic effects were negative for most analyses, as were those reported by Southwood and Kennedy (1990). However, for the all parity analysis of LWt and PWt the estimates were small and positive. No conclusions can be drawn from these correlations due to the small size of the m^2 effect, which indicates that the standard errors of the r_{am} estimates will be large.

Comparison of Large White and Landrace analyses:

Estimates of the heritability of gestation length,numbers born and weights at birth differ between the breeds. There are also small differences in the phenotypic means of these traits (table 5.6). The differences in means and variance components could be the result of selection or drift within the two breeds.

SUMMARY AND CONCLUSIONS

Estimates of heritabilities (c.0.1) and repeatabilities (c.0.2) for number born are in line with those found in the literature (Haley *et al.*, 1988). Estimates of common litter of birth effects and maternal genetic effects across all traits analysed were very low.

In conclusion, heritability and repeatability estimates in this population do not appear to be biased by maternal genetic effects and common family environmental effects, which can therefore be omitted from any breeding value estimation models. The reasons for the abscence of maternal genetic and common litter of birth effects may be the routine practice of cross-fostering and the masking of effects by environmental effects encountered after weaning. The precise effect of cross-fostering can not be quantified, but the results presented here may not be applicable in a population where cross-fostering is not in practice.

CHAPTER 6.

INDIVIDUAL ANIMAL MODEL ESTIMATION OF THE GENETIC CORRELATIONS BETWEEN PERFORMANCE TEST AND REPRODUCTION TRAITS AS RECORDED IN A COMMERCIAL LANDRACE POPULATION.

INTRODUCTION

Commercial selection programmes for pigs have concentrated mainly on the improvement of growth rates, lean content and feed conversion; reproduction traits of sows have been largely ignored. This is due to a number of factors: the low heritability of litter traits compared to growth and carcass traits, being able to record the traits on females only and having to wait until females are sexually mature before measurements can be taken. Also, a rise in the number of pigs produced per sow per year has been observed (Bichard *et al.*, 1983), although this improvement in pigs/sow/year may not be due to within breed selection but to improved husbandry and the use of crossbred sows in the commercial population (Haley *et al.*, 1988). However, Avalos and Smith (1987) showed that reasonable rates of genetic progress could be achieved for litter size, in particular if use was made of family information in an index. The most complete way of making use of all information available on relatives in selection decisions is through the use of Individual Animal Model BLUP to estimate the breeding values of all animals contained in the pedigree. This procedure assumes that all pedigree information and parameters are known without error, and while this criterion cannot be met, the use of Individual Animal Model REML within the population of interest provides the best estimates of the genetic

parameters.

Hill and Webb (1982) predicted that the relative economic value of selection for reproduction traits would increase as growth and carcass traits approach optimum values and progress in these traits becomes more difficult. Therefore, reliable estimates of the genetic correlations between reproduction and performance test traits are required for re-evaluation of breeding goals and making use of BLUP procedures.

Estimates of the genetic correlations between performance test and reproduction traits have been reviewed by Hill and Webb (1982) and more recently by Haley *et al.* (1988). The valid estimates ($-1 \leq r_g \leq +1$) given in table 1.4 for total number born with performance test traits vary from -0.15 to 0.44 for ADG, and from -0.54 to 0.62 for backfat depth, and generally have large standard errors.

The estimates to be found in the literature are from both commercial population analyses (Johannson and Kennedy, 1983; Löbke *et al.*, 1986) and selection experiments (de Nise *et al.*, 1983; Fredeen and Mikami, 1986). In the first cases, the sire half-sib covariances were estimated, and hence information from all relationships was not utilised; while in the selection experiments small numbers of observations were recorded. The analysis presented here is an individual animal model REML analysis, taking account of all known relationships and including records from all available sources.

MATERIAL AND METHODS

Data and population structure:

Performance test records

Performance test records were available on boars and gilts born and tested in a single nucleus herd over a six year period. Animals were selected for use as breeding stock on the basis of an index of performance test traits. The index contained ultrasonic backfat measurements, average daily gain on test and food conversion ratio on test for both males and females.

As recorded in chapters 2 and 3, boars and gilts underwent different performance tests. The feeding scale for gilts was lower than that for their male contemporaries throughout the data collection period; and gilts were penned in groups of ten, while boars were in pairs. Further details of the perfomance test can be found in chapter 2.

During the period of data collection, changes in the test regime and health status of the herd occurred which resulted in three distinct groups (Test Management Groups or TMGs) of data being identified within each sex. Briefly, the TMGs corresponded to periods when the animals were:- (a) on a low feeding scale; (b) on an increased feeding scale; and (c) exposed to pathogens (the health status of the herd was declining and the feeding scale was decreased during this period to minimise food wastage). The relative lengths of the periods corresponding to the three TMGs within each sex, and hence the relative size of each TMG in terms of quantity of data contained within them, were approximately 2:1:1. Details of these changes and the population structure within and across these groups are given in chapter 2.

In order that the maximum amount of information could be included in the analyses to estimate correlations between performance test and reproduction traits, performance test data were combined across all Test Management Groups. This assumes genetic correlations of one between traits as recorded in each TMG, within and between sexes.

In combining data across Test Management Groups, heterogeneity of variances and differences in mean performance need to be accounted for. The inclusion of month of test (defined in chapter 2 as four consecutive weekly batches of animals finishing test) within sex should remove differences in the mean level of performance due to environmental changes within a month of test, and as a result of this across Test Management Groups also. In order to standardise variances, the data were scaled by the residual standard deviation within Test Management Group, as estimated in the univariate analyses presented in chapter 2.

In all analyses reported in this chapter, the following Individual Animal Model was fitted to the performance test data:

$$Y_{ijklmn1} = a_{i1} + MT_j + b1.L_{ijkn} + b2.W_{ijln} + b3.D_{ijmn} + c_{n1} + e_{ijklmn1} \quad \text{(model 1)}$$

Where:

$Y_{ijklmn1}$ - is the phenotypic record of individual i for the trait;

a_{i1} - is the random additive genetic effect of the i^{th} individual;

MT_j - is the fixed effect associated with the j^{th} month of test within sex;

$b1.L_{ijkn}$ - is the partial regression of Y on length of test period (days);

$b2.W_{ijln}$ - is the partial regression of Y on weight at start of test (kg);

$b3.D_{ijmn}$ - is the partial regression of Y on age at start of test (days);

c_{n1} - is the random effect of the n^{th} litter of birth;

$e_{ijklmn1}$ - is the random environmental effect associated with the record, $Y_{ijklmn1}$.

The expectations of the additive direct genetic effect (a_{i1}), common litter of birth effect (c_{n1}) and residual error effect ($e_{ijklmn1}$) are zero, and their variances are σ^2_{a1}, σ^2_{c1} and σ^2_{e1} respectively. The covariances between a_{i1}, c_{n1} and $e_{ijklmn1}$ are zero.

Reproduction records

On selected females, reproduction records were available from either the nucleus or one of four multiplier herds. Up to five records were available on any sow, not necessarily beginning with the first parity or coming from consecutive parities. Details of the population structure and traits for reproduction data are given in chapter 5.

Haley *et al.* (1988) reviewed estimates of the genetic correlations between reproduction traits in different parities (litter size in particular), reaching the conclusion that making the assumption that these genetic correlations are one is reasonable. This assumption is inherent in the fitting of a repeatability model, which was done here in order to include as many records as possible in the bivariate analysis between the performance test and reproduction traits.

The Individual Animal Model fitted to the reproduction traits was:

$$Y_{ijkl2} = a_{i2} + s_{i2} + P_j + SBP_k + FHP_l + e_{ijkl2} \qquad \text{(model 2)}$$

Where:

Y_{ijkl2} - is the phenotypic record of sow i for the trait;

a_{i2} - is the random additive genetic effect of the i^{th} sow;

s_{i2} - is the permanent environmental effect of the i^{th} sow;

P_j - is the fixed effect of the j^{th} parity;

SBP_k - is the fixed effect of the k^{th} sow birth period (eight week periods);

FHP_l - is the l^{th} farrowing herd-farrowing period (eight week periods) combination;

e_{ijkl2} - is the random environmental effect associated with the record, Y_{ijkl2}.

The expectations of the additive direct genetic effect (a_{i2}), permanent environmental effect (s_{i2}) and residual error effect (e_{i2}) are zero, and their variances are σ^2_{a2}, σ^2_{s2} and σ^2_{e2} respectively. The three random effects are uncorrelated with each other.

Analyses performed:

In chapter 3 the analysis was performed using a bivariate algorithm proposed by Juga and Thompson (1990) which was an adaptation of the univariate Derivative-Free REML (DFREML) algorithm developed by Dr. K. Meyer (Meyer, 1988; Meyer, 1989a). This new algorithm estimated the genetic correlation between traits which had different fixed effect structures but which were not environmentally correlated. When considering analysis between performance test and reproduction traits it is necessary to be able to take account of:-

i. Different fixed effect structures - this was already included in the previous bivariate algorithm (see chapter 3, or Schaeffer, Wilton and

Thompson, 1978);

ii. Different random effect structures - a common litter of birth effect associated with performance test records and a permanent environmental effect on reproduction data were required. Visscher and Thompson (1992) had increased the number of random effects which could be fitted in the univariate case (previously limited to three), and only a slight further modification was required to correctly scale parameters according to the trait with which they were associated;

iii. Environmental correlation between traits - sows with reproduction records are also performance test recorded, there are therefore links between environmental effects on performance and reproduction traits, and an environmental correlation may be estimated.

The model for the bivariate analysis can be developed from the linear model for univariate analyses:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e} \qquad (3)$$

Equation (3) is a matrix representation of a general linear model where:

Y - is a vector of observations;

b - is a vector of fixed effects;

u - is a vector of random effects;

e - is a vector of random residual errors;

X,Z - are incidence matrices relating fixed and random effects to records.

The following (co)variance structure was assumed throughout:

$$V(\mathbf{u}) = \mathbf{G};$$

$$V(\mathbf{e}) = \mathbf{R};$$

$$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$$

$$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}.$$

The mixed model equations corresponding to this are then (Henderson, 1973):

$$\begin{bmatrix} \mathbf{X}'\mathbf{X} & \mathbf{X}'\mathbf{R}^{-1}\mathbf{Z} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{X} & \mathbf{Z}'\mathbf{R}^{-1}\mathbf{Z}+\mathbf{G}^{-1} \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{u}} \end{bmatrix} = \begin{bmatrix} \mathbf{X}'\mathbf{R}^{-1}\mathbf{Y} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{Y} \end{bmatrix} \quad \underline{\text{or}}\ \mathbf{C}\hat{\mathbf{f}} = \mathbf{r} \quad (4)$$

For a bivariate analysis between performance test and reproduction traits with different fixed and random effects associated with each trait (equations 1 and 2), equation (3) can be rewritten as:-

$$\begin{bmatrix} \mathbf{y}_1 \\ \mathbf{y}_2 \end{bmatrix} = \begin{bmatrix} \mathbf{X}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_2 \end{bmatrix} \begin{bmatrix} \mathbf{b}_1 \\ \mathbf{b}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{Z}_1 & \mathbf{0} & \mathbf{Z}_{c1} & \mathbf{0} \\ \mathbf{0} & \mathbf{Z}_2 & \mathbf{0} & \mathbf{Z}_{s2} \end{bmatrix} \begin{bmatrix} \mathbf{a}_1 \\ \mathbf{a}_2 \\ \mathbf{c}_1 \\ \mathbf{s}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{I}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{I}_2 \end{bmatrix} \begin{bmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \end{bmatrix}$$

Where:

$\mathbf{Y}' = \begin{bmatrix} \mathbf{y}_1' & \mathbf{y}_2' \end{bmatrix}$;

$\mathbf{X} = \begin{bmatrix} \mathbf{X}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_2 \end{bmatrix}$;

$\mathbf{Z} = \begin{bmatrix} \mathbf{Z}_1 & \mathbf{0} & \mathbf{Z}_{c1} & \mathbf{0} \\ \mathbf{0} & \mathbf{Z}_2 & \mathbf{0} & \mathbf{Z}_{s2} \end{bmatrix}$;

$\mathbf{b}' = \begin{bmatrix} \mathbf{b}_1' & \mathbf{b}_2' \end{bmatrix}$;

$\mathbf{u}' = \begin{bmatrix} \mathbf{a}_1' & \mathbf{a}_2' & \mathbf{c}_1' & \mathbf{s}_2' \end{bmatrix}$;

$\mathbf{e}' = \begin{bmatrix} \mathbf{e}_1' & \mathbf{e}_2' \end{bmatrix}$;

$\mathbf{X}_t, \mathbf{Z}_t, \mathbf{Z}_{Ct}, \mathbf{Z}_{St}$ - are incidence matrices relating fixed effects, random additive genetic, litter of birth and

permanent sow environment effects to records for trait t;

$\mathbf{I}_t$ - is an identity matrix with rank equal to the number of records on trait t;

$\mathbf{a}_t,\mathbf{c}_t,\mathbf{s}_t,\mathbf{e}_t,\mathbf{b}_t$ - are vectors of random additive genetic, litter of birth, sow environment, and residual effects and fixed effects for trait t, respectively;

1,2 - are subscripts to identify whether the vector/matrix is associated with performance test or reproduction records.

The variance-covariance structure is:-

$$V(\mathbf{u}) = \mathbf{G} = \begin{bmatrix} \mathbf{A}\sigma^2_{a1} & \mathbf{A}\sigma_{a12} & \mathbf{0} & \mathbf{0} \\ \mathbf{A}\sigma_{a12} & \mathbf{A}\sigma^2_{a2} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}_c\sigma^2_{c1} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}_s\sigma^2_{s2} \end{bmatrix};$$

$$V(\mathbf{e}) = \mathbf{R} = \begin{bmatrix} \mathbf{I}_1\sigma^2_{e1} & \mathbf{E}\sigma_{e12} \\ \mathbf{E}\sigma_{e12} & \mathbf{I}_2\sigma^2_{e2} \end{bmatrix}$$

$$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$$

$$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}.$$

Where:-

A - is the numerator relationship matrix;

$\mathbf{I}_c,\mathbf{I}_s$ - are identity matrices with rank equal to the number of levels of common litter of birth and permanent sow environmental effects, respectively;

E - is an incidence matrix relating performance test records to reproduction records.

The algorithm used in chapter 3 uses rescaling to simultaneously estimate two uncorrelated residual variances. In order that this may be carried out, the model can be reparameterised such that the two correlated residual effects are replaced by three uncorrelated effects. The linear model (equation (3)) is then represented as:-

$$\begin{bmatrix} \mathbf{y}_1 \\ \mathbf{y}_2 \end{bmatrix} = \begin{bmatrix} \mathbf{X}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_2 \end{bmatrix} \begin{bmatrix} \mathbf{b}_1 \\ \mathbf{b}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{Z}_1 & \mathbf{0} & \mathbf{Z}_{c1} & \mathbf{0} & \sigma^*_{e1}\mathbf{Z}_{r1} \\ \mathbf{0} & \mathbf{Z}_2 & \mathbf{0} & \mathbf{Z}_{s2} & \sigma_{e2}\mathbf{Z}_{r2} \end{bmatrix} \begin{bmatrix} \mathbf{a}_1 \\ \mathbf{a}_2 \\ \mathbf{c}_1 \\ \mathbf{s}^*_2 \\ \mathbf{r}^*_{12} \end{bmatrix} + \begin{bmatrix} \mathbf{I}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{I}_2 \end{bmatrix} \begin{bmatrix} \mathbf{e}^*_1 \\ \mathbf{e}_2 \end{bmatrix}$$

Where $\mathbf{Z}_{r1}, \mathbf{Z}_{r2}$ are incidence matrices relating residual covariance effects to records on performance test and reproduction records respectively, and $\mathbf{r}^*_{12}$ is a vector of residual covariance effects. The superscript * indicates an effect which has changed interpretation relative to the basic bivariate model.

The residual covariance effect is coded as applying to all records on an individual, regardless of which trait is recorded. In this way, animals with no reproduction records (boars and unselected gilts) had a unique level associated with their single test record while sows had the same level across test and reproduction records. In considering this coding, it is clear that the covariance estimated from this effect will be partitioned from the permanent environmental variance of the sow for reproduction traits, since it acts across parities ($\mathbf{Z}_{r2} = \mathbf{Z}_{s2}$), and the residual variance for performance test traits (since $\mathbf{Z}_{r1} = \mathbf{I}_1$).

The variance-covariance structure for the reparametrised model is:-

$$V(\mathbf{u}) = \mathbf{G} = \begin{bmatrix} \mathbf{A}\sigma^2_{a1} & \mathbf{A}\sigma_{a12} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{A}\sigma_{a12} & \mathbf{A}\sigma^2_{a2} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}_c\sigma^2_{c1} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}_s\sigma^{*2}_{s2} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}_1\sigma^*_{r12} \end{bmatrix};$$

$$V(\mathbf{e}) = \mathbf{R} = \begin{bmatrix} \mathbf{I}_1\sigma^{*2}_{e1} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}_2\sigma^2_{e2} \end{bmatrix}$$

$$\text{cov}(\mathbf{u},\mathbf{e}') = 0;$$

$$V(\mathbf{Y}) = \mathbf{ZGZ}' + \mathbf{R}.$$

Scaling is carried out by the algorithm such that $\sigma^*_{e1}=\sigma_{e2}=1$ and the scaled genetic (co)variance matrix, $\mathbf{G}^*_a$, is $\Sigma^{-1/2}\mathbf{G}_a\Sigma^{-1/2}$, where:

$$\Sigma = \begin{bmatrix} \sigma^{*2}_{e1} & 0 \\ 0 & \sigma^2_{e2} \end{bmatrix} \quad \therefore \quad \Sigma^{-1/2} = \begin{bmatrix} 1/\sigma^*_{e1} & 0 \\ 0 & 1/\sigma_{e2} \end{bmatrix}$$

$$\mathbf{G}_a = \begin{bmatrix} \sigma^2_{a1} & \sigma_{a12} \\ \sigma_{a12} & \sigma^2_{a2} \end{bmatrix}$$

Which gives:

$$\mathbf{G}^*_a = \begin{bmatrix} \sigma^2_{a1}/\sigma^{*2}_{e1} & \sigma_{a12}/\sigma^*_{e1}\sigma_{e2} \\ \sigma_{a12}/\sigma^*_{e1}\sigma_{e2} & \sigma^2_{a2}/\sigma^2_{e2} \end{bmatrix}$$

In chapter 3, a single common environmental effect was estimated across both traits (that is, a perfect correlation was assumed between effects on both traits), and it was expressed relative to one of the traits. In these analyses, the common litter of birth and permanent environmental effects are associated with one trait only. In order that all variances are correctly scaled and transformation back to the original scale is

straightforward, they are scaled by the appropriate residual variances. Similarly, the residual covariance effect, which is expressed in both traits, is scaled by the product of the residual standard deviations of the two traits. After scaling **G** and **R** are:

$$\mathbf{G} = \begin{bmatrix} \mathbf{A}\otimes\mathbf{G}_a^* & 0 & 0 & 0 \\ 0 & I\sigma_c^2/\sigma_{e1}^{*2} & 0 & 0 \\ 0 & 0 & I\sigma_s^{*2}/\sigma_{e2}^2 & 0 \\ 0 & 0 & 0 & I\sigma_{e1e2}/\sigma_{e1}^*\sigma_{e2} \end{bmatrix}; \quad \mathbf{R} = \begin{bmatrix} I\sigma_{e1}^{*2} & 0 \\ 0 & I\sigma_{e2}^2 \end{bmatrix}$$

$$\mathbf{G}^{-1} = \begin{bmatrix} \mathbf{A}^{-1}\otimes\mathbf{G}_a^{*-1} & 0 & 0 & 0 \\ 0 & I\sigma_{e1}^{*2}/\sigma_c^2 & 0 & 0 \\ 0 & 0 & I\sigma_{e2}^2/\sigma_s^{*2} & 0 \\ 0 & 0 & 0 & I\sigma_{e1}^*\sigma_{e2}/\sigma_{e1e2} \end{bmatrix}$$

$$= \begin{bmatrix} \mathbf{A}^{-1}\otimes\begin{bmatrix} \alpha_1 & \alpha_{12} \\ \alpha_{12} & \alpha_2 \end{bmatrix} & 0 & 0 & 0 \\ 0 & I\lambda_c & 0 & 0 \\ 0 & 0 & I\lambda_s & 0 \\ 0 & 0 & 0 & I\lambda_r \end{bmatrix}$$

$$\mathbf{R}^{-1} = \begin{bmatrix} I1/\sigma_{e1}^{*2} & 0 \\ 0 & I1/\sigma_{e2}^2 \end{bmatrix} = \begin{bmatrix} I\gamma_1 & 0 \\ 0 & I\gamma_2 \end{bmatrix}$$

Where:-

$\otimes$ is the direct (Kronecker) product as defined in Searle (1966);

$$\begin{bmatrix} \alpha_1 & \alpha_{12} \\ \alpha_{12} & \alpha_2 \end{bmatrix} = \mathbf{G}_a^{*-1} = \phi \begin{bmatrix} h_2^2/e_2^2 & -h_{12}/e_1e_2 \\ -h_{12}/e_1e_2 & h_1^2/e_1^2 \end{bmatrix};$$

h_1^2, h_2^2 are the heritabilities of the traits in males and females;

e_1^2, e_2^2 are the residual variances as a proportion of the phenotypic variances;

h_{12} is the coheritability, $\sigma_{a12}/\sigma_{p1}\sigma_{p2}$, (Yamada, 1968) of the two traits;

ϕ $= e_1^2 e_2^2 / (h_1^2 h_2^2 - h_{12}^2)$.

The numerator relatationship matrix , **A**, for each Test Management Group was based on all available pedigrees going back to the beginning of the data collection period (that is, those associated with the earliest records in Test Management Group 1). By substitution into (4), the mixed model equations for the analysis can be derived.

Approximation of maximum likelihood estimates:

Because the combined reproduction and performance test data set was very large (9342 animals with 8961 test records and 5291 reproduction records) and the method used required coding for residual covariance effects (therefore increasing the number of equations by 8961), the time required for each likelihood evaluation was high. It was decided not to iterate each analysis to a solution, but to define a grid of points relative to the two covariance parameters of interest and fit a quadratic surface through the likelihoods evaluated under the assumption that other variance components would remain at their univariate maximum likelihood estimates.

The bivariate algorithm used estimates the phenotypic variance in each trait as $\sigma_{ee}^2/(1-\sum_{i=1}^{np}\theta_i)$; where np is the number of (co)variance parameters, θ_i are (co)variance parameters and σ_{ee}^2 is the estimate of the residual variance. The residual covariance parameter, r_{12}, is $\sigma^*_{r12}/\sigma^*_{p1}\sigma^*_{p2}$. Expectations of true

phenotypic variances from variance components estimated under this bivariate algorithm and univariate analysis are:

Trait:	Bivariate:	Univariate:
1	$\sigma^2_{a1} + \sigma^2_{c1} + k_1.\sigma^{*2}_{p1}.r_{12} + \sigma^{*2}_{e1}$	$\sigma^2_{a1} + \sigma^2_{c1} + \sigma^2_{e1}$
2	$\sigma^2_{a2} + \sigma^{*2}_{s} + k_2.\sigma^{*2}_{p2}.r_{12} + \sigma^2_{e2}$	$\sigma^2_{a2} + \sigma^2_{s2} + \sigma^2_{e2}$

Where k_1 is the ratio $\sigma^*_{e1}\sigma^*_{p2}/\sigma^*_{p1}\sigma_{e2}$, k_2 is the reciprocal of k_1. The factors k_1 and k_2 account for the scaling of the residual covariance component. Equating terms in these equations gives $\sigma^2_{e1} = \sigma^{*2}_{e1} + k_1.\sigma^{*2}_{p1}.r_{12}$ and $\sigma^2_{s2} = \sigma^{*2}_{s} + k_2.\sigma^{*2}_{p2}.r_{12}$.

For each analysis a grid of nine points, consisting of all combinations of three values for each covariance parameter, was assessed, the likelihood function being evaluated at each point, with all other parameters at their approximated bivariate (scaled univariate) values. Approximate values of all (co)variance components were calculated from the above equations, and parameter estimates generated from these.

The likelihood function evaluated was (Meyer, 1989a):

$$-2 \log L = \kappa + \log |\mathbf{R}| + \log |\mathbf{G}| + \log |\mathbf{C^*}| + \mathbf{y'Py} \qquad (5)$$

Where κ is a constant term, $\mathbf{C^*}$ is a full rank submatrix of the coefficient matrix (that is, $\mathbf{C}$ of equation 4 with $\mathbf{X}$ replaced by a full rank submatrix $\mathbf{X^*}$) and $\mathbf{P}$ is a matrix such that:

$$\mathbf{P} = \mathbf{V}^{-1} - \mathbf{V}^{-1}\mathbf{X^*}(\mathbf{X^{*\prime}V}^{-1}\mathbf{X^*})^{-1}\mathbf{X^{*\prime}V}^{-1} \quad ; \text{and } \mathbf{Y} \sim N(\mathbf{Xb}, \mathbf{V})$$

The log determinant of $\mathbf{C^*}$ was accumulated by the algorithm during the Gaussian elimination step and the other components of the likelihood were calculated as:

$$\log |\mathbf{R}| = (n_{rec1} - n_{f1})\,\sigma^{*2}_{e1} + (n_{rec2} - n_{f2}) \log \sigma^{2}_{e2}$$

$$\log |\mathbf{G}| = n_{anim}\left\{ \log \frac{\sigma^{2}_{a1}}{\sigma^{*2}_{e1}} + \log \left(\sigma^{2}_{a2} - \frac{\sigma_{a1a2}\sigma_{a1a2}}{\sigma^{2}_{a1}} \right) \frac{1}{\sigma^{2}_{e2}} \right\}$$
$$+ n_c \log \frac{\sigma^{2}_{c1}}{\sigma^{*2}_{e1}} + n_{sow} \log \frac{\sigma^{*2}_{s}}{\sigma^{2}_{e2}} + n_{rec1} \log \frac{\sigma_{e1e2}}{\sigma^{*}_{e1}\sigma_{e2}}$$

$$\mathbf{y'Py} = \frac{ypy_{11}}{\sigma^{*2}_{e1}} + \frac{2ypy_{12}}{\sigma^{*}_{e1}\sigma_{e2}} + \frac{ypy_{22}}{\sigma^{2}_{e2}}$$

Where:

n_{anim} - is the number of animals in the analysis;

n_{reci} - is the number records for trait i;

n_{fi} - is the total numbers of fixed effect levels for trait i;

n_c, n_{sow} - are the number of levels of common litter of birth and permanent sow environment random effects;

ypy_{jk} - are the elements of **y'Py**

The likelihood value produced by the algorithm can be rescaled so that the all variance components are at their univariate values rather than at approximations of these by replacement of terms in bivariate variance components with equivalent terms in univariate variance components. The following quadratic function with respect to the two covariances was then fitted through the likelihoods evaluated:

$$\mathbf{L} = \alpha + \mathbf{X'b} + \mathbf{X'BX}$$

Where **L** is a vector of nine likelihood function values evaluated at each point in the 3x3 grid, **X** is a 2x9 matrix containing all combinations of the covariance parameters used to define the grid, and the constants contained in **b** and **B** are unknown. The values of **b**, **B** and α were estimated using matrix algebra procedures given in Searle (1966). By

differentiation of the quadratic function with respect to **X**, and setting the differential to zero (for a maxima or minima), the maximum likelihood values for x can be estimated thus:

$$\frac{d\mathbf{L}}{d\mathbf{X}} = \mathbf{b} + 2\mathbf{B}\mathbf{X}$$

$$\text{if } \frac{d\mathbf{L}}{d\mathbf{X}} = 0, \text{ then: } \quad \mathbf{X}_{max} = -0.5\mathbf{B}^{-1}\mathbf{b}$$

Where $\mathbf{X}_{max}$ is a vector containing the maximum likelihood estimates of the two covariance parameters.

If the maximum likelihood estimate of either covariance parameter fell outside the area specified by the grid, the procedure was repeated with new covariance parameter estimates.

Similarly, a new grid was specified if the approximate variance-covariance matrix of the covariance parameters $\{(-2\mathbf{B})^{-1}\}$ was negative definite. This tended to happen if the dimensions of the grid were wide, the quadratic relationship breaking down the further from the maximum likelihood values the parameters were (a ridge was observed, indicating correlation between the covariance parameters). As a result of this, most analyses were run at least twice, once with the dimensions of the grid quite wide, and once with the grid points set close around the first approximations. Despite repeat running, the quadratic approximation procedure was still expected to be faster than downhill simplex iteration, because of the large number of parameters involved.

The univariate estimates required for this method came from chapter 5 in the case of reproduction traits, and from analysis under model 1 for performance test data combined across Test Management Groups. The method used in the analysis of the performance test data was the same as

analyses under model 2 in chapter 2.

In order to check the effectiveness of this method, two bivariate analyses between ADG and PWt were performed, one by quadratic fitting and the other by downhill simplex iteration until the variance of the function values (-2 times the log likelihood) stored in the simplex was less than $1x10^{-7}$. The results from the simplex iteration analysis given in table 6.5 can be compared with those from the quadratic fitting procedure which appear in tables 6.3 and 6.4.

RESULTS

Phenotypic means of performance test traits are given in table 2.4 (chapter 2), within Test Management Groups, and for reproduction traits in table 5.6 (chapter 5).

Results from univariate analysis of the performance test data combined across Test Management Groups as described are in table 6.1, while the univariate analysis results of Number Born Alive (NBA) and Average Piglet Weight (PWt) from chapter 5 are reproduced in table 6.2.

The analysis was limited to four performance test traits, ADG, ADFI, FCR and C Fat. The other traits (weight gain on test and three measures of fat depth) were omitted because they were highly correlated with traits in the analysis.

The parameter estimates for the performance test traits across all Test Management Groups are similar to the mean of the estimates produced in chapter 2 for individual Test Management Groups within sexes. These results plus those in table 6.2 were used as starting values for the analysis

between performance test and reproduction traits.

Table 6.1: Results of analysis of Landrace performance test data, both sexes across all test management groups. Data scaled by approximate residual variance within each Test Management Group.

Trait	σ_a^2	σ_c^2	σ_e^2	σ_p^2	h^2	c^2
C Fat	0.632	0.080	1.04	1.75	0.36	0.05
ADFI	0.193	0.086	1.02	1.30	0.15	0.07
ADG	0.188	0.073	1.10	1.37	0.14	0.05
FCR	0.262	0.070	1.10	1.43	0.18	0.05

Table 6.2: Results of analysis of Landrace reproduction data (for full details of analysis, see chapter 5).

Trait	σ_a^2	σ_s^2	σ_e^2	σ_p^2	h^2	s^2	r
NBA	0.776	0.685	6.32	7.79	0.10	0.09	0.19
PWt	0.0111	0.00367	0.0373	0.0521	0.21	0.07	0.28

Tables 6.3 and 6.4 contain the covariance and correlation estimates respectively. The approximate standard error of the covariance estimates, calculated as the square root of the diagonal elements of $(-2\mathbf{B})^{-1}$, is included in table 6.3. The standard errors of the correlation estimates were approximated from the sampling variances of the covariance estimates (the squares of the standard errors) assuming that the variance components are known without error, and are given in table 6.4.

In the final runs of the quadratic fitting method, the dimensions of the grid with respect to the genetic and environmental covariance could be summarised as a percentage of the standard error of the final estimate for the covariance in question. The dimensions were between 83% and 173%

and between 87% and 121% for the genetic and environmental covariances, respectively.

The estimates of the genetic correlation ranged from -0.052 to 0.082, with the estimates for C Fat, ADFI and FCR with NBA, and of ADFI and ADG with PWt being positive. The largest absolute value of the genetic correlation of a performance test trait with a reproduction trait was 0.082 (FCR with NBA).

Environmental correlation estimates ranged from -0.028, for C Fat with NBA, to 0.059, for C Fat with PWt.

Table 6.5 contains the results from a bivariate analysis of ADG with PWt allowing every parameter to vary within the usual parameter space. The Simplex iterative procedure was used to find the maximum likelihood values of all the parameters. The analysis was converged when the variance of the seven highest values of -2 times the log-likelihood encountered was less than 1×10^{-7}. There is very little difference in the estimates of the variance components for each trait compared to the univariate analysis. Both the genetic and environmental correlation estimates have the opposite sign to their counterparts from quadratic fitting with variances fixed at their univariate values, but the estimates are of low magnitude.

Table 6.3: Estimates of genetic and residual covariances of NBA and PWt with selected performance test traits.

Test trait	Reproduction trait			
	NBA		PWt	
	σ_{a1a2}	σ_{e1e2}	σ_{a1a2}	σ_{e1e2}
C Fat	0.1438 (0.081)*1	-0.0719 (0.076)	-0.0044 (0.011)	0.0116 (0.010)
ADFI	0.0789 (0.058)	-0.0174 (0.062)	0.0005 (0.006)	-0.0047 (0.006)
ADG	-0.0320 (0.052)	-0.0036 (0.067)	0.0028 (0.006)	-0.0012 (0.006)
FCR	0.1049 (0.058)	0.0222 (0.076)	-0.0051 (0.006)	-0.0045 (0.007)

*1 - standard error of covariance estimate, from the second derivative of the quadratic equation (see text), zero values are due to rounding errors;

Table 6.4: Estimates of genetic and residual correlations of NBA and PWt with selected performance test traits.

Test trait	Reproduction trait			
	NBA		PWt	
	r_g	r_e	r_g	r_e
C Fat	0.072 (0.116)	-0.028 (0.030)	-0.028 (0.131)	0.059 (0.051)
ADFI	0.071 (0.150)	-0.007 (0.024)	0.006 (0.130)	-0.024 (0.031)
ADG	-0.029 (0.136)	-0.001 (0.025)	0.033 (0.131)	-0.006 (0.030)
FCR	0.082 (0.129)	0.008 (0.029)	-0.052 (0.111)	-0.022 (0.035)

*1 - approximate standard error of correlation assuming variances known without error

Table 6.5: Results of bivariate analysis of ADG with PWt. Simplex iterative procedure used to locate maximum likelihood with no fixed parameters.

1. Variance components and parameters

Trait	σ_a^2	σ_c^2	σ_s^2	σ_e^2	σ_p^2	h^2	c^2	s^2
ADG	0.186	0.0720	-	1.11	1.36	0.14	0.05	-
PWt	0.0112	-	0.0036	0.0373	0.0520	0.22	-	0.07

2. Covariance components and parameters

σ_{a12}	r_g	σ_{e12}	r_e
-0.0002	-0.005	0.0055	0.027

DISCUSSION

In order that the maximum amount of information could be included in the analyses to estimate correlations between performance test and reproduction traits, data were combined across all Test Management Groups. In doing this it was assumed that there was no genotype x environment interaction between the Test Management Groups. While the analyses to test this assumption within sex have not been perfomed within this population, Cameron *et al.* (1988) found there to be no evidence of sire x feeding regime interactions in a selection experiment where animals were assigned to either completely *ad libitum* feeding or restricted feeding, expected to be a far more extreme situation than any comparisons possible in this population. There are no reported estimates of genotype x health environment interactions, although Kennedy and Quinton (1987) reported differences in genetic and environmental levels between herds with

different health classifications in Canada. They pointed out, however, that the genetic differences could have arisen because of differences in selection pressure applied by the farmers in response to the different environmental effects observed, a situation whch would not have occurred here, since the period in question is too short for selection effects to be great.

The high estimates of the genetic correlations between performance test traits as recorded in the two sexes given in chapter 3, support the assumption made in combining male and female test data that these genetic correlations are one.

In chapter 2 it was observed that in the second and third boar Test Management Groups the inclusion of the common litter of birth effect did not significantly affect the fit of the model ($p>0.01$; using a likelihood ratio test to compare models with and without a common litter of birth effect included). This may have been due to either compensatory feeding in these two groups (which were the least restricted of any of the TMGs) overcoming the common litter of birth effect, or an inability to estimate the parameter (there were a high proportion of litters with only a single representative, which was expected to make partitioning of the effect more difficult).

By including a common litter of birth effect for all performance test traits, the explaination that data structure caused the inability to detect a significant common litter of birth effect in the last two boar TMGs is assumed to be true. A unit correlation between common litter of birth effects on male and female litter mates was assumed, implying that combining the data will have increased the accuracy of our evaluation of the c^2 parameter.

No common litter of birth effect was included in the model for reproduction traits. The univariate analyses of reproduction traits reported in chapter 5 indicated that this effect is of no importance in these traits, the parameter estimates being of very low magnitude.

The genetic correlation estimates produced in this study confirm the conclusions drawn in previous reports, that number born is weakly correlated with performance test traits (Hill and Webb, 1982) and the correlation between backfat and NBA is unfavourable (positive) (Johannson and Kennedy, 1983).

Löbke *et al.* (1986) produced both paternal half-sib and daughter-dam estimates of the genetic correlations between growth traits and reproduction traits, concluding that the daughter-dam estimates may have been biased by maternal effects. In chapters 2 and 5, it was shown that no maternal genetic effect could be detected for either performance test or reproduction traits in this population, and that no common environmental effect of litter of birth could be detected for reproduction traits. Therefore, having fitted common litter of birth as a random effect associated with performance test traits in this analysis, it was expected that the results would be similar to the paternal half-sib estimates produced by Löbke *et al.* (1986). The genetic correlation estimate for NBA with C Fat (0.07) is in close correspondence with the first litter estimate of Löbke *et al.* (0.03),but is considerably lower than the estimate produced for Number Born over the first three litters (NB3; r_g = 0.28). This may be because the NB3 value is the sum over these litters, and hence is confounded with the permanent environmental effect of the sow.

The growth rate traits analysed by Löbke *et al.* (1986) also differ in

their definition from those studied here (Löbke *et al.* analysed 12 and 24 week weights), and so the estimates produced by them may not be directly comparable with those given here. For Number Born in the first litter a low negative genetic correlation with 12 week weight (-0.32) and a zero estimate with 24 week weight were produced (standard errors between 0.15 and 0.30), whilst for ADG with NBA a very low negative genetic correlation estimate was produced here (-0.029).

Simplex iterative bivariate analysis of ADG and PWt gave estimates of genetic and environmental covariances which were opposite in sign to those from the quadratic fitting procedure (comparison of table 6.5 with table 6.3). Estimates from both sources were of low magnitude. The genetic covariance estimate from the Simplex iterative procedure was within one standard error unit of the quadratic fitting estimate, while the environmental covariance was just outside of this range.

Comparison of the variance components from the iterative bivariate analysis of ADG with PWt (table 6.5) with their univariate counterparts (tables 6.1 and 6.2) shows that the bivariate estimates were between 97.3% and 100.9% of the univariate estimates. This supports the decision to fix the parameter estimates at their univariate values.

The genetic correlations produced were generally unfavourable, that is selection upon one of the traits in the accepted desired direction would produce a correlated selection response in the other trait in the undesirable direction. For example, long term selection to reduce C Fat would also reduce NBA and PWt. The sizes of the correlations are such, however, that the effects upon reproduction traits of selection on performance test traits taking no account of correlations with reproduction traits would not be seen

for many generations. The genetic correlations vary from zero by a maximum of 0.64 standard error units (for the correlation between NBA and FCR) and are therefore unlikely to be significantly different from zero.

SUMMARY AND CONCLUSIONS

Bivariate estimates of genetic and environmental correlations between reproduction traits (NBA and PWt) and growth traits (C Fat, ADFI, ADG and FCR) of Landrace pigs were calculated. The data were analysed across sexes and periods corresponding to differences in the test and health regimes.

The estimates were produced using a quadratic approximation to estimate the maximum likelihood values with respect to the covariance parameters, while all other parameters were fixed at their univariate estimates.

For all combinations of performance test traits with reproduction traits the resulting correlation estimates were low. This indicates that:-

i. There will have been little effect upon reproduction traits from the rigorous selection carried out upon performance test traits over the years;

ii. when incorporating reproduction data into BLUP analysis procedures it would be possible to analyse performance test and reproduction traits separately.

As a result of ii. it should be possible to make savings on computer resources and time taken for the analysis of all traits.

CONCLUDING REMARKS.

As the use of multivariate BLUP to estimate breeding values for pigs becomes more widespread, reliable estimates of genetic parameters for the traits of interest are required. However, the results of the present study indicate how greatly these parameters can change with the environment. In this population the major environmental changes were due to management and disease.

Where the environment changes due to health, or any other factor outwith managment control, the effects upon the levels of variation in the population can only be observed retrospectively. However, for management changes aimed at improving the genetic performance of the population the subsequent effect upon genetic and phenotypic variation, and hence upon breeding value estimates and rates of genetic gain should be considered. It may be that changes in the heritability, as observed in this study, are predictable, at least for certain types of management change such as going from restricted to *ad libitum* feeding.

In the bivariate analysis of performance test data with reproduction data, some assumptions were made with regard to both performance test and reproduction traits. The model fitted was the most plausible model which it was feasible to fit given the computer intensive nature of the work. Advances in computer power and statistical methods may allow more complicated models to be fitted in the near future. Of the assumptions made in chapter 6 the most important ones to test are the unit genetic correlations assumed between traits recorded in different TMGs within sex and between reproduction traits in nucleus and muliplier herds.

It is possible to test the assumption that a unit genetic correlation between TMGs within and across sexes for any trait by carrying out a series of analyses of the type performed in chapter 3 (to estimate the genetic correlation between traits with no environmental covariance, see Schaeffer, Wilton and Thompson ,1978). Genetic correlation estimates between traits as recorded in different TMGs within a sex would give an indication of the size of any genotype-environment interaction between the TMGs. Given the consistently high correlations between traits as recorded in males and females (chapter 3), where the environments varied more than any within sex between TMG comparison, this exercise was not carried out.

Similarly, the presence of genotype-environment interaction for reproduction traits between nucleus and multiplier levels could be tested. It may be that there is no genotype-environment interaction for reproduction traits across breeding pyramid levels due to similarities between sow housing and management in the two levels.

The use of the quadratic fitting procedure to estimate genetic correlations between performance test and reproduction traits reduced the amount of computing involved in these analyses. In doing this variance components were fixed at their univariate maximum likelihood estimates, which will have restricted the possible values of the covariances to some extent. A study of the likelihood surface, to reveal whether or not a quadratic relationship between the likelihood and the covariance components existed, and tests of the effect of fixing the variance components at values other than their univariate estimates would be of interest.

The Individual Animal Model takes account of selection provided all information is included in the analysis. Given that selection was performed

on an index of performance test traits, all analyses which do not include at least the index traits will be biased by selection. However, it is expected that this bias will be less under the IAM than under other analytical models due to making use on information from all relationships.

Heritability estimates for performance test traits in chapter 2 tended to be low when compared to literature estimates. This may reflect some population specific environmental factor not accunted for in the model fitted. Alternatively, it could be a result of selection and the structure of the data set. The pedigree information available does not go beyond the parents of the earliest recorded animals in the data set (which were born in 1982). Since this population had been under selection for a number of years prior to this, the base animals in our analysis are a selected group with the reduced genetic variance associated with this.

Heritability and repeatability estimates for number born and number born alive agree with those in the literature. Given the low genetic correlations found between performance test and reproduction traits in chapter 6, these estimates should not be biased by the exclusion of data on the selection index traits.

Estimation of the genetic correlations between performance test traits was carried out as a series of bivariate analyses using the method of Thompson and Hill (1990), although this method was capable of a full multivariate analysis. Improvements could be made to the Thompson and Hill algorithm as incorporated into the univariate derivative-free REML programs of Meyer (1989a) to make it more robust to low variance estimates and high correlation estimates, and therefore more able to perform a multivariate analysis on any data set with equal design matrices.

The bivariate algorithm utilised in chapter 6 was not available at the time the correlations between the performance test traits were being estimated.

Fowler *et al.* (1976) proposed that FCR should be included in the selection criteria for populations under controlled feeding, since it had no antagonistic effect upon food intake in their study. Gu (1988) produced estimates of the genetic correlation between FCR and ADG of -1.0, and concluded that there was no point in including FCR in the selection objective for that population, which was on a very restricted feeding scale. The estimates in this study vary from -0.14 to -0.93 across TMGs and sexes. These correlations indicate that none of the groups of animals analysed in this study were fully restricted and that the approach of Fowler *et al.* (1976) should be used, with selection index weights reappraised when changes are made to the management system.

Given the almost zero estimates of the genetic correlations between performance test and reproduction traits from chapter 6 for this population incorporation of reproduction traits into the selection goal, with the aims being to increase number born alive and average piglet weight, would not conflict with the selection aims for performance test traits. It would therefore be possible to produce a single line selected for both improved performance test and reproduction traits. Two points should be made with regard to such a line:-

1. progress in any given trait will be lower than in a line selecting on fewer traits;

2. Nelson et al. (1990) observed a reduction in the number born alive in a line selected for extreme low backfat, it is possible that antagonistic genetic correlations between performance test and reproduction traits

would be produced as selection proceeded.

Any decision to form a single selection line incorporating both performance test and reproduction traits in the selection criteria would have to take account of the above factors. Genetic progress in either performance test traits or reproduction traits will be faster and antagonistic genetic correlations induced are not important in specialised lines. However, if all resources used in maintaining two specialised lines were made available to a single line, the increased selection differentials possible may outweigh the loss of selection pressure in the index on any given trait.

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